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INHIBITION OF CARBONIC ANHYDRASE IN THE TREATMENT OF GLAUCOMA

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In his Proctor Award Lecture on the formation of the intra-ocular fluid, Friedenwald [1] expressed the view that more effort might profitably be directed toward a reduction in the formation of aqueous in addition to the present approach which concerns itself largely with an effort to increase the outflow from the eye. His work has inspired a useful advance in the therapy of glaucoma.

In the pre-war years our ideas on the formation of the aqueous were dominated by Leber and Starling's views of the importance of osmotic and diffusional forces in the transfer of water and solutes across the capillary wall. Studies of the relative chemical composition of the aqueous and the blood plasma led Duke-Elder,[2] at first, to the conclusion that the composition of the aqueous resembled that of a dialysate of the blood plasma.

The use of more refined techniques showed that this view could no longer be maintained and it seemed probable that electrolytes were transferred into the eye by a secretory process. The secretory organ appeared to be the ciliary body. Friedenwald's work [3] led him to postulate that the main product of this 'gland' would be a slightly hypertonic fluid whose chief electrolyte was bicarbonate. The reaction by which this is effected is $OH + CO_2 \rightarrow HCO_3$.

Kinsey [4] showed that there is, in fact, a considerable excess of bicarbonate in the aqueous of the rabbit's eye as compared with the blood, and he ascribed to it a pilot role in the formation of the intra-ocular fluid. In a later paper, Kinsey [5] demonstrated that the concentration of the bicarbonate in the posterior chamber exceeded that in the anterior chamber in the proportion of 100 : 81. This supported the idea that the bicarbonate was formed by the ciliary body.

The conversion of water and carbon dioxide into carbonic acid is known to be catalyzed by the enzyme carbonic anhydrase. It was therefore of some interest to determine whether this enzyme was present in the ciliary region of the eye. Wistrand [6] was able to prove that carbonic anhydrase is present in the ciliary processes and iris of the rabbit.

From the foregoing it seemed likely that the enzyme carbonic anhydrase played an important part in the formation of the aqueous. As a corollary, it was logical to enquire whether inhibition of this catalyst would reduce the formation of aqueous. An inhibitor of carbonic anhydrase in the form of the heterocyclic sulphonamide acetazoleamide (Diamox) had been known for some time [7] and was thought to have a low toxicity. Preliminary trials with rabbits by Grant and Trotter [8] showed that the intravenous injection of Diamox produced a definite lowering of the intra-ocular pressure.

The idea of using Diamox in glaucoma seems to have occurred at about the same time to a number of American ophthalmologists. The pioneer publication on the subject was that of Becker.[9] He found Diamox a useful agent in lowering the intra-ocular pressure in man. Becker used single oral doses of 500-1,000 mgms. Grant and Trotter [8] considered Diamox to be a safe and helpful drug in the treatment of certain types of glaucoma. They published a chart showing the fall in intra-ocular pressure produced by Diamox in a series of 45 human eyes. The decrease in tension observed may best be described by the boxing maxim that the higher they come the harder they fall.

The present writers have had the opportunity of observing the effects of Diamox in a number of cases of glaucoma. In those reported below Diamox proved helpful where miotic therapy alone was ineffective in reducing the intra-ocular pressure.

CASE REPORTS

Case 1. Mr. M.D., aged 77, developed glaucoma in the left eye after needling of an after-cataract on 8 April 1954. The raised tension was not sufficiently lowered by eserine or by cortisone used

locally. On 6 August the tension in mm. Hg (Schiotz) was as follows: right eye 32, left eye 60. The patient was given 125 mg. of Diamox t.d.s. He discontinued the eserine on his own initiative because it was beginning to irritate his eye. On 13 August the tension was: right 27, left 30. He was now put on pilocarpine 1% and the Diamox in the same dosage. On 27 August he complained that the tablets were causing gastric discomfort. The ocular tension was now right 25, left 28. On the same treatment his ocular tension was maintained at the same level for another fortnight. The Diamox was then discontinued. On 24 September the tension was right 19, left 25, and the corrected visual acuity in the left eye was 6/12 part, where it had been 6/36 at the commencement of treatment.

Case 2. Mrs. E.V.A., aged 54. Thrombosis of central vein in left eye, April 1953. The eye was blind. When seen on 20 August 1954 the tension of the left eye was 60 (Schiotz). She was put on Diamox, 125 mg. t.d.s. On 27 August the tension in this eye was 15. No miotics were used.

Case 3. Mrs. T., aged 60, developed acute congestive glaucoma after the dislocation of a hypermature cataract. D.F.P. used for 4 days failed to reduce the tension below 70. She was then given 500 mg. of Diamox 6-hourly for 3 days, followed by 125 mg. 6-hourly for 3 days more. The tension was reduced to 30 and the eye was then operated on. This patient complained of paraesthesia in the course of Diamox therapy.

Case 4. A male patient with Marfan's syndrome developed acute glaucoma following upon the dislocation of his left lens into the anterior chamber. He was in acute pain. The condition was not adequately controlled by standard miotic therapy. He was then put on Diamox, which produced a prompt reduction in the ocular tension and allowed operation to be performed on the eye with safety.

DISCUSSION

The exhibition of carbonic anhydrase inhibitor (Diamox) proved useful in the cases of glaucoma described. The only toxic symptoms noted were paraesthesias in one case and gastric discomfort in another. Acetazoleamide would appear to be safe when given for short periods, but in view of the wide distribution of the enzyme carbonic anhydrase in the human body one would be hesitant about using it in large doses for long periods in the chronic forms of glaucoma. Tonographic studies by Becker and by Grant and Trotter show that there is no improvement in the facility of outflow of aqueous when Diamox is used. This seems to indicate that the drug acts by reducing the rate of formation of the aqueous. Further confirmation of this possibility is found in the suppression of the water provocative test in glaucoma by Diamox.[10] The diuretic effect of the drug does not appear to be of importance in glaucoma. Diuretics, as such, have not proved of value in glaucoma therapy.

SUMMARY

The authors describe 4 cases of glaucoma in which Diamox proved of value. They believe it to be a useful adjunct to existing methods of therapy in glaucoma.

Thanks are due to Dr. M. Franks and the Superintendent of the Johannesburg Hospital for permission to publish details of cases 1 and 2 and to Dr. E. Miller for details of case 4.

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CONGRESS RESOLUTIONS

The Secretary of the Association has received the following letter dated 1 February 1955 from the private secretary of the Minister of Health.

Dear Sir,

With further reference to your letter dated 20 September 1954 I am directed by the Honourable the Minister of Health to reply as follows to the two resolutions adopted at the 39th S.A. Medical Congress:

1. *Notification of Cancer*

Representations have previously been made to Dr. van Rhijn, former Minister of Health, to the effect that cancer should be declared a notifiable disease in terms of the Public Health Act, 1919, as amended. This matter is at present being investigated by the Department of Health.

2. *Milk and its Products*

The State has already done much in the direction advocated by the Congress as the following indicate:

Milk powders form part of the enriching mixtures used for the enrichment of both bread and mealie meal. In the latter case the scheme is still in its infant stage and is conducted largely as an experiment.

Under the State-aided Milk Scheme fresh milk, or full-cream powdered milk, when fresh milk is not available, is supplied free of charge, through pre-school milk clubs usually controlled by Welfare Organisations, to European, Coloured and Asiatic children of pre-school age. The scheme is limited to children of this age-group only, as children of school-going age participate in the National School-Feeding Scheme, which is administered by the various Provincial Administrations.

The following quantities of fresh and full-cream powdered milk have been distributed during the past three years, free of charge, to pre-school children, viz.:

	Fresh Milk	*Powdered Milk*
1951/52	220,000 gallons	9,000 lbs.
1952/53	214,000 gallons	9,000 lbs.
1953/54	188,500 gallons	12,600 lbs.

In addition to the above, the following quantities of condensed milk and powdered milk were distributed at economical prices by the mobile markets of the State Food-Distribution Scheme which operate in the sub-economic areas of the larger cities, viz.:

	Condensed Milk	*Powdered Milk*
1951/52	1,217,800 lbs.	4,546 lbs.
1952/53	1,231,900 lbs.	13,039 lbs.
1953/54	1,156,700 lbs.	12,300 lbs.

During times of surpluses in production, the Department of Nutrition, when requested to do so by large distributors in Pretoria, undertakes to sell buttermilk and skimmed milk at a reduced price in the Native locations.

Under the State-aided Margarine and Butter Scheme, margarine and butter are sold through the mobile markets of the State Food Distribution Scheme in the sub-economic areas of the larger cities, and through margarine and butter committees in the rural areas, to the sub-economic groups at a very much reduced price.

The Department of Nutrition has also now commenced with the distribution, through the State Food-Distribution Scheme, of cheese at a subsidised price.

Yours faithfully,
(Signed) H. J. Dreyer,
Private Secretary.

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VAN DIE REDAKSIE

ENSIEME IN TERAPIE

'n Aantal ensieme is waardevolle hulpmiddels met die mediese en chirurgiese behandeling van sekere kondisies. As hul in gesuiwerde vorm versigtig en met oorleg gebruik word, is dit onwaarskynlik dat hul 'n nadelige uitwerking kan hê. Onder hierdie ensieme sorteer die proteolitiese ensieme soos tripsien en die verbinding van streptokinase en streptodornase (SK–SD). 'n Tweede groep, verteenwoordig deur hialuronidase, wat maklik in 'n relatief suiwer vorm verkry kan word, depolimeriseer hialuroniese suur, die grondstof van weefsels.

Kristallyne tripsien is 'n sterk proteolitiese middel wat in 'n neutrale medium (optimum pH 8·0) effektief is. Dit verteer nekrotiese weefsel en proteïene, behalwe fibrien, en word in die vorm van vloeistof of poeier vir wonde gebruik in hoeveelhede wat van die grootte van die wond afhang; gewoonlik word met ongeveer 50 mg. begin maar daarna kan die hoeveelhede geleidelik tot 400 mg. verhoog word. In die plasma is 'n aktiewe anti-tripsien aanwesig wat met aansteeklike en ander siektes toeneem. Die nadele verbonde aan die gebruik van tripsien is dat dit irriterent mag wees, verswakte weefsels mag wegvreet en reaksies mag uitlok as gevolg van die absorbering van proteose; laasgenoemde reaksies kan waarskynlik deur anti-histamienmiddels beheer word. 'n Klein hoeveelheid tripsien binneaars ingespuit het 'n skielike en noodlottige gevolg gehad.

'n Verbinding van streptokinase (in ensiem-aktiveermiddel) en streptodornase maak fibrien, asook sekere selbestanddele, vloeibaar en is uiters waardevol met die behandeling van wonde. 'n Oplossing van 100,000 eenhede SK en ongeveer 25,000 eenhede SD in 10-20 ml. is vir 'n wond geskik en as die wond besonder suur of alkalies is kan van 'n bufferoplossing gebruik gemaak word. Plasmafaktor (plasminogeen of serum profibrienolisien) beïnvloed hul werking en die aktiwiteit van die SK–SD wissel na gelang die hoeveelheid plasmafaktor in die deurgesygde stof. Die koorsreaksies wat hierdie preparaat mag veroorsaak as dit op groot rou oppervlaktes of in toe spasies gebruik word, kan waarskynlik aan die aanwesigheid van pirogene toegeskryf word.

Vir die débridement van 'n wond of 'n brandwond kan SK–SD oplossing of tripsien daarop gespuit word of as nat verbande aangewend word. Vir diep wonde of holtes kan 'n kateter gebruik word en as die holtes bedek is kan 'n inspuiting gegee word mits dat alle voorsorg getref word om enige gevaar van 'n inspuiting in die long of bloedstroom te vermy. Tripsien verrig uitstekende

EDITORIAL

ENZYMES IN THERAPY

A number of enzymes are proving valuable adjuncts to the medical and surgical treatment of certain conditions. It appears that in their purified form they are not likely to do harm if used with care and understanding. Amongst the enzymes thus used in therapy are proteolytic enzymes such as trypsin, and the combination of streptokinase and streptodornase (SK–SD). A second group, represented by hyaluronidase, which is readily available in a relatively pure form, depolymerize hyaluronic acid, the intercellular or ground substance of the tissues.

Crystalline trypsin is a strong proteolytic agent, effective in a neutral medium (optimum pH 8·0), which will digest necrotic tissue and proteins other than fibrin, and is used for that purpose in wounds. It is applied as liquid or powder, the amount depending on the size of the wound; ordinarily about 50 mg. would be used in a first application but the amount may be gradually increased to 400 mg. There is in the plasma an active anti-trypsin which becomes increased in infectious and other diseases. Disadvantages in the use of trypsin are that it may prove irritating, it may possibly erode devitalized tissues, and it may produce reactions from the absorption of proteoses; antihistamine drugs may control these latter reactions. A small amount of trypsin injected intravenously has caused sudden death.

Streptokinase (an enzyme-activator) and streptodornase in combination bring about the liquefaction of fibrin as well as certain cellular elements, and are of great value in the treatment of wounds. A solution containing 100,000 units SK and approximately 25,000 units SD in 10-20 ml. may be used on a wound, and in a buffer solution if the wound is very acid or very alkaline. Their activity is affected by plasma factor (plasminogen or serum profibrinolysin) and the activity of the SK–SD varies according to the amount of plasma factor in the exudate. The febrile reactions that may be produced by this preparation used in closed spaces or on large raw surfaces appear to be due to the presence of pyrogens in the material.

For debridement of a wound or burn SK–SD solution or trypsin may be applied by spray or as a wet dressing. In deep wounds and sinuses the solution may be intro-

diens met die ensiemagtige débridement van koue absesse, sere en wonde in die beginstadium asook om verdikte afskeidings in die asemhalingskanaal vloeibaar en los te maak. SK–SD is besonder waardevol vir die oplossing van gestolde borsbloeding, verdikte empieem-vloeistof, bloedblase in die urineblaas of elders en vir absesse, wonde en sere wat plasminogeen bevat.

Streptokinase aktiveer plasminogeen. Dit is al in 'n onsuiwer vorm uit mens- en beesplasma verkry en mag binnekort vir débridement en moontlik vir die oplossing van binnespierse trombose beskikbaar wees. Plasmien-profibrienolisien, wat deur SK–SD geaktiveer is, het proefondervindelik effektief klonte wat deur natrium morruaat veroorsaak is, opgelos.

Hialuronidase maak die tussenselstof vloeibaar deur die hialuroniese suur te depolimeriseer en dit help om die stowwe in die selle te diffundeer. Hierdie ensiem kan van baie bronne verkry word, vernaamlik van beesteelballe. Dit is beskikbaar in die vorm van 'n droë poeier wat stabiel en maklik oplosbaar is. Die werking word gemeet in troebelheidvermindering-eenhede (TRU) of viskositeitvermindering-eenhede (VRU) en 1 TRU staan gelyk aan 3·3 VRU. Die werking word deur verskeie faktore beïnvloed. Hialuronidase word dikwels met onderhuidingspuitings gebruik om die absorbering van vloeistowwe te bespoedig, dit is veral handig met die behandeling van babas wanneer binneaarse terapie moeilik is. 'n Enkel dosis kan nou of gedurende die beginstadium van die inspuiting gegee word of saam met die inspuiting. As gevolg van die snelle absorbering bestaan daar dieselfde gevare en teenaanduidings as in die geval van baie vinnige inspuitings. Op dieselfde manier word hialuronidase gebruik om die absorbering te verhoog van middels soos kontrasmiddels en geneesmiddels soos penicillin en heparin. Dit word ook gebruik met die spalking van beenbreuke; in oogheelkunde, om die verspreiding van plaaslike verdowingsmiddels aan te help en om die opblaas en vervorming van weefsels te verminder, en om die herabsorbering van bloed en vloeistof soos bv. in pretibiaal-miksedeem en parafimose, aan te moedig.

Die terapeutiese gebruik van ander ensieme word ondersoek en die geleentheid vir navorsing oor hierdie besonder belangrike en interessante onderwerp is groot. 'n Volledige verslag oor die huidige posisie met byna 300 verwysings na literatuur oor die onderwerp het onlangs verskyn.[1]

1. Cliffton, E. E. (1954): Amer. J. Med. Sci., **228**, 568.

duced by means of a catheter. It may be applied in closed cavities by injection, when care must be taken to avoid any danger of injection into the lung or the blood stream. Trypsin has proved most useful in the enzymatic debridement of early chronic abscesses, ulcers and wounds, and also for the liquefaction and clearing of thick secretions in the respiratory tract. SK–SD is most useful in the lysis of clotted haemothorax, thickened empyema fluid, haematomas in the urinary bladder or elsewhere, and in abscesses, wounds and ulcers containing plasminogen.

Plasminogen is activated by streptokinase. It has been obtained in impure form from human and bovine plasma, and may soon be available for use in debridement and possibly for lysis of intravascular thromboses. Plasmin—profibrinolysin activated by SK–SD—has proved effective experimentally in the lysis of clots produced by sodium morrhuate.

Hyaluronidase liquefies the intercellular substance by depolymerization of hyaluronic acid, and thus aids the diffusion of materials to the cells. The enzyme is available from many sources, especially the bovine testis. It is available as a dry powder, which is stable and readily soluble. The activity is expressed in turbidity-reducing units (TRU) or viscosity-reducing units (VRU) and 1 TRU is approximately equal to 3·3 VRU. The activity is influenced by numerous factors. A common use of hyaluronidase is to increase the speed of absorption of fluids in hypodermoclyses; it is particularly useful in infants, in whom intravenous therapy is difficult. A single dose may be given before or during the early part of the clysis, or mixed with the clysis. Because of the rapid absorption there are the same dangers and contra-indications as with very rapid infusions. A similar use of hyaluronidase is the enhancement of absorption of agents such as contrast media and drugs like penicillin and heparin. It is also used in the reduction of fractures; in ophthalmology, to increase the diffusion of local anaesthetics and to reduce ballooning and distortion of the tissues; and to promote the reabsorption of collections of blood and fluid, e.g. in pretibial myxoedema and in paraphimosis. Many other uses have been suggested for this enzyme.

The therapeutic use of other enzymes is being developed and there is much scope for research in this important and interesting field. A full account of the present position has been published recently with nearly 300 references to the literature on the subject.[1]

1. Cliffton, E. E. (1954): Amer. J. Med. Sci., **228**, 568.

UNION DEPARTMENT OF HEALTH BULLETIN

Report for the 7 days ended 3 February 1955:

Plague, Smallpox: Nil.

Typhus Fever. Cape Province: One (1) Native case in the Herschel district. One (1) Native case in the Glen Grey district. Diagnosis of both cases confirmed by laboratory tests. *Transvaal:* Three (3) Native cases in the Heidelberg district. Diagnosis confirmed by laboratory tests.

Epidemic Diseases in other Countries:

Plague: Nil.

Cholera in Chittagong, Dacca (Pakistan).

Smallpox in Mergui, Moulmein (Burma); Phnom-Penh (Cambodia); Bombay, Lucknow, Madras, Tellicherry (India); Karachi, Lahore (Pakistan); Mogadiscio (Somalia); Tanga (Tanganyika).

Typhus Fever in Yosu (Korea).

RESPIRATORY ALLERGY IN THE COASTAL AREAS OF SOUTH AFRICA

THE SIGNIFICANCE OF CLIMATE

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Symptoms may be precipitated and maintained in a person with an allergic tendency by exogenous factors such as inhalants, ingestants and physical influences as well as by endogenous factors including bacterial and parasitic infections and endocrine and psychological disturbances. These possible trigger factors operate singly or in combination and their identification is desirable in the simplification of the therapeutic approach to the allergic patient.

In respiratory allergy of the seasonal type the inhalation of the prevailing pollens is the factor responsible. In the perennial or non-seasonal variety pollens do not play a part and other agents must be sought.

Seasonal respiratory allergy in South Africa has been described [1] in detail but certain aspects with a present bearing will be referred to here. Grass pollinosis is the only seasonal respiratory allergy of importance and occurs in summer from October to March with a maximum incidence in December and January. The limitation of symptoms to this period characterizes the condition and renders etiological diagnosis simple.

Seasonal pollinosis in South Africa occurs mainly but not exclusively in the natural grasslands of the country (Fig. 1). These grasslands are largely confined to the Northern Karroo or Highveld (C in Fig. 1)—the central plateau which covers most of the Transvaal, particularly in its southern part, the Orange Free State, and the western portion of the Eastern Cape Province. This plateau, 4,000-6,000 feet above sea-level is situated in the region of spring and summer rainfall with long cold dry winters.

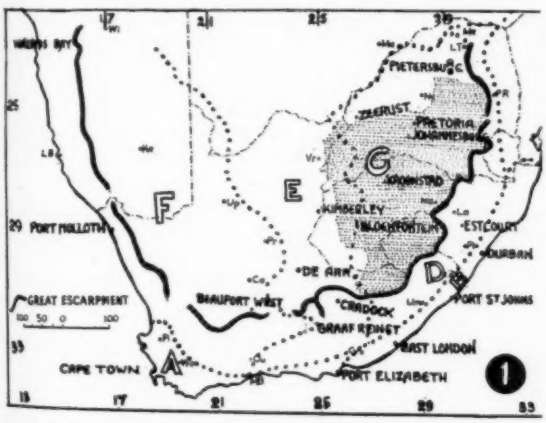

Fig. 1. Map of the Union of South Africa showing the principal inland and coastal towns as well as the Great Escarpment. The dotted lines divide the country into the following regions: A—South-West Cape, B—coastlands of South-East Cape and Natal, C—Eastern Plateau, D—Eastern Plateau slopes, E—Semi-arid Interior, F—deserts. The Natural Grasslands are indicated in the stippled area.

The greater portion of the Union of South Africa has an elevation of some 3,000 feet above sea-level, the area below 1,500 feet consisting of a narrow fringe around the coast. The vegetation of the coastal region extending east and north-east and including Port Elizabeth, East London and Durban (B in Fig. 1) is of the temperate savanna and sub-tropical forest types with comparatively little grass. In the South-West Cape (A in Fig. 1), in which Cape Town is the principal city and which has a winter rainfall, the vegetation is of the sclerophyl type. Much of the territory on the west of South Africa consists of semi-desert vegetation with hardly any grass. In the adjoining temperate savanna areas there is open bush with an undergrowth of grass.

Perennial respiratory allergy, which includes allergic vasomotor rhinitis and sinusitis (nasal and paranasal allergy) as well as bronchial asthma, is of common occurrence in South Africa and often presents a difficult etiological and therapeutic problem.

Bronchial asthma could hardly be overlooked by any physician but the allergic conditions of the upper respiratory tract are often incorrectly regarded as of infective origin and dealt with by chemotherapeutic, antibiotic and operative measures. To an even greater extent the minor respiratory allergies, including recurrent and continuous 'colds' and constantly blocked or running nose, are frequently considered of trifling significance. The neglect of these conditions is to be deplored, for they may be the precursors of more serious allergic states and demand adequate treatment and control. The confident prophecy of the physician that 'the child will grow out of it' sometimes comes true but not infrequently is unwarranted optimism.

Perennial respiratory allergy is both clinically and etiologically no different in South Africa from elsewhere, and the recognized exogenous and endogenous causative factors are frequently found; but there remains a considerable proportion of cases where careful clinical and laboratory investigations fail to reveal specific factors. It is especially amongst such cases that the etiological significance of climate has been studied.

Attention was drawn in 1951 [2, 3] to the fact that the incidence of perennial respiratory allergy is comparatively high at the coast, especially the East Coast, where the condition of sufferers from inland areas tends to become worse. Extensive experience of patients in different parts of the country since that time has amply confirmed this view. Large numbers of patients have been investigated in this connection in Johannesburg and at the larger coastal towns of the Union. The

geographical relationship of these towns to one another and to the Great Escarpment, where land surfaces rise to higher levels from the coast, is shown in Fig. 1. It is our opinion that climate *per se* is a significant exciting or trigger factor in the disturbance of the equilibrium which an allergic subject may be enjoying.

The cases described below, typical of many patients seen in Johannesburg, represent people reasonably well-balanced allergically in the Highveld of the Transvaal and Orange Free State but in whom symptoms of bronchial asthma or allergic rhinitis developed or were significantly aggravated when they lived at or visited the coast.

PATIENTS SEEN IN JOHANNESBURG

Case 1. Mr. W., clerk, aged 28 years. East Rand. Suffered from bronchial asthma in Port Elizabeth where he lived until 18 years of age. He had only 2 attacks of asthma in his army service, which included Central Africa, Italy and Burma. On his return he visited Port Elizabeth for 2 weeks and suffered from asthma all that time. In Johannesburg, where he then lived for more than 7 years, he was free from symptoms. He developed asthma only on visiting the coast and for a few days after his return home.

Case 2. Mrs. H., aged 27 years. Johannesburg. Suffered from recurrent 'colds' in East London up to the age of 7 years, when asthma commenced. Came to Johannesburg when 10 years old and had considerable vasomotor rhinitis but very little bronchospasm. She is fairly well at Cape Town but, on a recent visit to Durban developed asthma severe enough to warrant hospital treatment there for a month.

Case 3. Miss W., nurse, aged 21 years. Johannesburg. Born in Germiston and lived in Durban from 1940 to 1950, where bronchial asthma commenced 4 years after arrival. In Johannesburg the asthma gradually subsided and she then suffered mainly from 'hay fever'. On a subsequent visit to Durban she developed severe asthma a day after arrival and was compelled to return to Johannesburg. She had attacks of asthma in Durban on 2 subsequent visits, one in summer and one in autumn.

Case 4. Miss S., clerk, aged 37 years. Lives in Johannesburg where she was born. Vasomotor rhinitis commenced at the age of 15 years while on holiday in East London and has continued since then in Johannesburg but in milder form. Each March in subsequent years she has visited Durban or a South Coast resort and on each occasion developed asthma. During a vacation at Cape Town on account of severe asthma she was compelled to return to Johannesburg, where her chest cleared within a week. She enjoyed good health on vacations in the Drakensberg and in Rhodesia but has had 2 attacks of asthma subsequently in Johannesburg.

Case 5. Mr. F., student, aged 18 years. Johannesburg. Suffered from bronchial asthma for 18 years while living in Port Elizabeth. He had no symptoms in Bloemfontein, where he lived from 1945 to 1948. There was some recurrence of symptoms in Johannesburg but in milder form. He is always worse at the coast, particularly Beira, Durban and Port Elizabeth.

Case 6. Mrs. S., aged 22 years. Johannesburg. Suffered from vasomotor rhinitis during most of her school days. Her condition improved as she grew older and she was quite free of symptoms for a year before her marriage. She visited Durban on honeymoon and asthma commenced 2 days after arrival there and continued throughout a coastal voyage from Durban to Cape Town. On the return journey she became free of symptoms in the Karroo and remained well in Johannesburg. She later visited Margate, Natal South Coast, and suffered from asthma, which however disappeared in Johannesburg. On a subsequent visit to Port Elizabeth asthma recurred for more than 2 weeks but again she had no further symptoms in Johannesburg.

Case 7. Miss N., clerk, aged 18 years. Benoni. Suffered from 'colds' since childhood. For the last 3 years has had attacks of vasomotor rhinitis and asthma lasting a day or two, which occur every few weeks but not related to menstruation. She was notably worse at the Natal South Coast towns of Doonside and Margate, where she had to seek medical advice on each occasion. In February 1954 severe asthma commenced a fortnight after arrival on vacation in Scottburgh, also on the South Coast, and continued for the remaining week of her holiday there.

Case 8. Mrs. L., aged 28 years. Johannesburg. Vasomotor rhinitis in Johannesburg from the age of 9 years. Lived in Durban for 7 years from the age of 20 and suffered from bronchial asthma throughout that period. On her return to Johannesburg a year ago she was very much improved, with only occasional mild bronchospasm and some 'hay fever'. She developed severe asthma while on a 3-weeks vacation visit to Durban recently.

Case 9. v. W., European girl, aged 6½ years. Germiston. The details are graphically illustrated in Fig. 2. She was born in East London and from a few months old suffered from constant 'colds' until 10 months old, when the family moved inland. For more than 2 years at Maritzburg and Johannesburg she was completely free of symptoms. She again moved to East London and remained well for 10 months, when she commenced to suffer from asthma, which rapidly increased in severity and then continued, in a somewhat milder form at first, on moving inland again to Germiston. It is interesting to speculate whether asthma would have occurred at all had the patient not returned to East London.

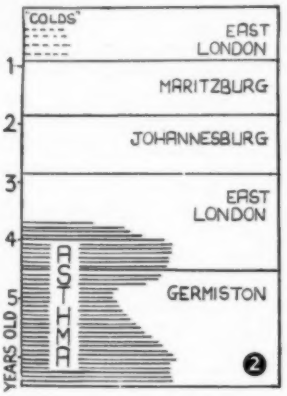

Fig. 2. *Case* 9. Child v.W. (see text). The chart demonstrates the commencement of symptoms of respiratory allergy in infancy at the coast, their absence in inland areas and their recurrence in severe form on return to the coast. Amelioration of symptoms no longer occurs inland.

Case 10. Mrs. S., Johannesburg. The details are illustrated graphically in Fig. 3, and show how a visit to Margate, Natal, apparently precipitated asthma in a woman who had not previously suffered from respiratory allergy. It will be observed that there was an exacerbation of symptoms each time she paid a vacation visit to the Natal South Coast and that the patient has not so far regained her original good health.

Case 11. V., a married native woman aged 26 years, was born and lived in Zeerust, where she was always quite well, not even subject to 'colds'. She obtained employment in Johannesburg in 1948 and for 5 years was quite well. In 1953, as a domestic servant she accompanied her mistress and family for a few weeks holiday in Scottburgh on the Natal South Coast. During her stay there she developed vasomotor rhinitis, which has persisted in Johannesburg for more than a year. This case demonstrates the fact that coastal conditions may precipitate respiratory allergy in non-Europeans also.

Exacerbation of symptoms in an allergic person in a new environment may, of course, have nothing to do with climate but may be related to new specific allergens encountered or to new circumstances associated with greater stress conditions. In studying the case histories of allergic persons whose symptoms developed or became worse at the coast special note was taken of such possible allergenic and psychological factors. These however may be ruled out in the generality of cases on the simple proposition that sufferers tend to become worse at the coast whereas the inland movement of such patients from the coast is nearly always followed by improvement in allergic health.

Alleviation of a patient's symptoms in another area may be due to the absence of pollens or fungi to which he is sensitive. As already indicated specific pollen

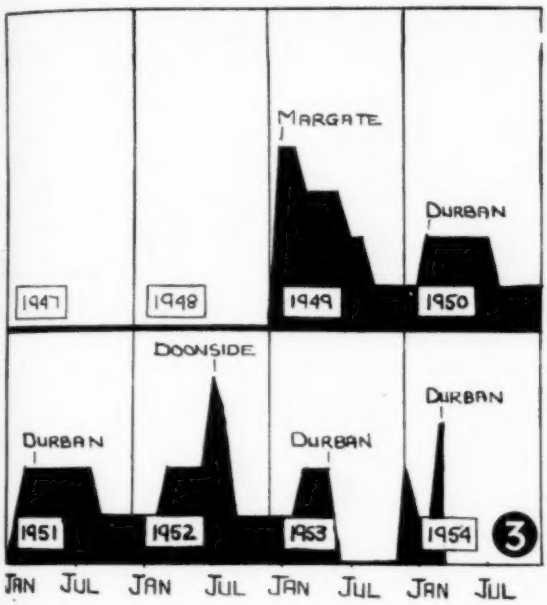

Fig. 3. *Case* 10. Mrs. S. (see text). The chart demonstrates the initiation and exacerbation of symptoms of respiratory allergy in the coastal towns indicated.

factors can be significant only where symptoms are limited to the seasons in which the plants flower. Seasonal hay fever due to grass pollens does not infrequently occur at the coast, but is readily differentiated by its characteristic summer incidence. In Durban and northward where the pollens of the lush subtropical vegetation are frequently suspect, pollen sensitization cannot be important, because the heavy pollens of tropical plants are insect-borne and in any event would produce pollinosis only in the season of their flowering. In Port Elizabeth, Cape Town and certain parts of Natal, where acacia trees are abundant, there is a widely-expressed opinion that the exacerbation of symptoms in spring is due to the pollens of these trees, which flower from about August to October. Specific skin tests, however, have not confirmed this view. It is possible that the dry powdered flowers when blown about by the wind produce non-specific irritation of the nasal and bronchial mucosa. Similar exacerbation of symptoms in the autumn so frequently complained of cannot of course be accounted for by sensitivity to acacia pollen. As respiratory allergy at the coast is of the perennial type and because skin tests with local pollens do not confirm this possibility a pollen etiology must be discounted.

The warmth and humidity of the subtropical areas on the eastern shores of Southern Africa suggest that atmospheric fungi might play a part in local respiratory allergy. Householders there often complain of moulds on clothing, leather goods and foodstuffs in the home. Numerous cases selected for us by physicians and otorhinolaryngologists as typical of local perennial bronchial asthma and allergic rhinitis were submitted to fungus skin-sensitivity tests in Durban, Port Elizabeth and Cape Town with a wide range of fungus allergens. Sensitivity was demonstrated only in a small proportion of cases and in no greater numbers than in some of the inland cities of the Union where similar studies have been made. Exposure of plates of culture media to the atmosphere in these coastal towns did not reveal the presence of fungus elements different from those found in Johannesburg, where atmospheric fungus studies have been carried out for many years. The fungus content of house dust and bedding materials from the homes of sufferers at the coast was not found to be significantly different from corresponding material in Johannesburg. A fungus factor therefore in coastal respiratory allergy has so far not been substantiated.

It has been suggested that in damp atmospheres house dust is rendered more allergenic by the action of micro-organisms [4] or that it is quantitatively increased by the breaking-down action of fungi on animal or plant fibres. [5] These views still await scientific confirmation. Investigation to this end is being pursued in our laboratories but large numbers of allergic patients both from inland and coastal areas have shown similar skin reactions to extracts of house dust derived from either region. An enhanced *clinical* sensitivity to coastal house dust is a possibility and this aspect is being studied.

PATIENTS SEEN IN THE COASTAL TOWNS OF SOUTH AFRICA

Perennial respiratory allergy was also studied in towns on the South Coast and East Coast of South Africa from Cape Town to Lourenco Marques and including Port Elizabeth, East London and Durban. Lourenco Marques is just outside the borders of the Union and will therefore not be especially considered here but, with its high incidence of vasomotor rhinitis and bronchial asthma presents the same picture as in the coastal areas further south.

Numerous patients were investigated at each of these coastal towns and the cases described hereunder are examples of those in whom significant amelioration of symptoms occurred when they moved inland.

Cape Town

Respiratory allergy of the perennial type is commonly seen in Cape Town and occurs in Europeans and Eurafricans (Coloured) but to a lesser extent in Natives. The incidence of bronchial asthma is greatest in the spring (September and October) and in autumn (April). The climate is relatively humid in March and April and fogs occur in the low-lying parts of the city especially in the Cape Flats. Respiratory allergy is invariably worse in damp or windy weather, and its incidence is thought by some to be relatively high in the Cape Flats, Pinelands and Milnerton areas. Bronchial asthma and nasal allergy commonly affects Natives and Coloured people who visit Cape Town from their homes in the country.

Case 1. Miss G., nurse, aged 34 years. Was born in Namaqualand where she spent most of her life and was quite well. Asthma

commenced in Cape Town after a few years residence there. She is quite well during vacations at home.

Case 2. Mrs. S., aged 43 years. Quite well before she came to live in the Cape Flats, where she has suffered from asthma for 15 years. She has no symptoms on her visits to the Karroo.

Case 3. Mr. R., train conductor, aged 29 years. Asthma for 21 years in George. Much improved when he lived in the Transvaal for 7 years. Vasomotor rhinitis and bronchial asthma commenced within 14 days of his arrival in Cape Town and have persisted.

Case 4. Mrs. G., Native housewife, aged 36 years. At her home in Burghersdorp was always quite well. Arrived in Cape Town in 1941 and after a year began to suffer from bronchial asthma, which is becoming worse.

Case 5. Mrs. N., Native nurse, aged 27 years. Quite well at her home in Queenstown. Vasomotor rhinitis commenced shortly after her arrival in Cape Town. The condition cleared up during vacation in Grahamstown but recurred almost at once on her return to Cape Town.

Case 6. Native nurse, aged 34 years. Her home is in Cala in the Transkei, where she is always quite well. Bronchial asthma commenced when she arrived in Cape Town in 1949 and has persisted.

Port Elizabeth

Perennial respiratory allergy is very common in Port Elizabeth. The incidence is highest in the spring (August to October) and in autumn (March to May), when the weather is wet and windy. The areas near the sea front have a worse reputation than those on a somewhat higher level. The exacerbation of the condition in spring is ascribed by many to the pollen of the acacia trees, which flower at that time. Europeans and Coloured people suffer with equal frequency and severity but Natives appear to be less affected. Doctors agree that nearly all sufferers improve in health when they move inland, mainly to Graaff Reinet, Cradock, Cookhouse and the Swartberg and Suurberg regions.

Case 1. Miss W., aged 16 years. Well to the age of 4 years and thereafter suffered from recurrent 'colds'. Asthma for the last 3 years in Uitenhage near Port Elizabeth. She is always quite well in Cradock, Graaff Reinet and Kommadagga.

Case 2. Miss G., Coloured, aged 28 years. Bronchial asthma for the last 9 years. Commenced a few years after her arrival in Port Elizabeth from Graaff Reinet, where she was always quite well.

Case 3. Miss E., Coloured, clerk. Suffered from vasomotor rhinitis for 4 years in Port Elizabeth. The condition cleared completely while on a visit to Graaff Reinet but recurred on her return to Port Elizabeth.

Case 4. Mr. E., aged 49 years. Vasomotor rhinitis commenced some 17 years ago, on arrival in Port Elizabeth from Johannesburg, where he had been free from respiratory complaints. On a visit to Johannesburg in July 1952 the condition cleared up completely but recurred on his return to Port Elizabeth.

Case 5. Master H., 6 years. Born in Port Elizabeth. He was perfectly well until whooping cough occurred at the age of 2½ years. For six months thereafter he suffered from head and chest 'colds' followed by bronchial asthma. He has a watery nasal discharge every morning and a constant post-nasal drip except on very fine days. He is worse in the afternoons and evenings 'when the wind comes up'. In each of a number of 3-4 week periods spent in Oudtshoorn he was quite well. He was somewhat wheezy' in Cape Town on 3 weeks holiday there.

East London

Perennial respiratory allergy is very common in East London and is said to be aggravated when the East winds blow from the sea. By others, however, the exacerbation of symptoms is ascribed to the pollen of the acacia trees which flower from August to October. Similar exacerbations however also occur in February and March. Symptoms are worse at the beach areas and patients very often improve in health when moving to suburbs on a higher level. Almost invariably patients are relieved of their symptoms in Cradock, Queenstown and Stutterheim and even as near as Kei Road some 20 miles inland. The incidence is high in Natives who however recover completely in the Transkei or Ciskei but whose symptoms inevitably reappear on their return. Natives from Lovedale where they are quite well frequently complain that they do not enjoy a symptom-free night in East London.

Case 1. P., European boy, aged 7 years. Asthma commenced at the age of 2 years in East London after an attack of whooping cough. He is quite well at Tarkastad. On returning to East London he remains free from symptoms for a month or two only before the asthma recurs.

Case 2. Mrs. v. d. M., aged 22 years. Asthma since the age of 7 years in East London. The condition improves at Queenstown and other inland places. Her symptoms are as bad in Durban and Cape Town as in East London.

Case 3. Coloured, ward-maid, aged 60 years. Had lived in East London since 1937. After a vacation at her home in Mooiplaas some 20 miles distant she returned to East London in 1942. Within a few weeks asthma developed and has persisted. She is well at Mooiplaas and also at Fort Beaufort, about 19 miles inland.

Case 4. E., Native male, aged 53 years. Has always been quite well at his home in Sterkstroom. He commenced suffering from asthma after 4 years in East London. He was free of symptoms during a visit of a month to Sterkstroom during 1953 but the condition recurred almost immediately on his return.

Durban

Perennial respiratory allergy is very common in Durban in Europeans, Africans and Indians. The climate is subtropical in character, especially in the summer, and characterized by high temperature and high relative-humidity.

Case 1. Mr. B., aged 34 years. Allergic rhinitis commenced in Durban in 1941 and bronchial asthma in 1945. He is quite well during visits to Johannesburg and Kimberley.

Case 2. Mr. M., aged 43 years. Asthma since 1940 in Durban. Quite free of symptoms in the Transvaal and in Barkly East.

Case 3. Mr. K., storeman, aged 19 years. Bronchial asthma commenced 3 weeks after his arrival in Durban from Vryheid, where he has always been quite well.

Case 4. Mrs. J., aged 23 years. Hay fever since childhood in Johannesburg, where the incidence was seasonal and hardly troublesome. The condition is worse in Durban, where asthma occurs, mainly at night.

Case 5. P., Indian male, aged 15 years. Constant 'colds' for the last year. Was quite well for 6 weeks in Johannesburg, but the condition recommenced on his return to Durban.

Although opinion at the coast is unanimous that the perennial type of bronchial asthma and nasal allergy occurs with greater frequency and often with greater severity than in inland areas, there is by no means agreement as to the factors responsible. Indeed, the explanations offered are varied and often conflicting. Sufferers themselves sometimes assign causes for the periodic exacerbation of symptoms and blame weather, winds, rain or pollen. There is little disagreement however in the view that the majority of patients benefit on moving to the interior. The distance from the coast where such improvement occurs varies with different patients but, in general, improvement is noticed, and sometimes dramatically,

at higher levels towards the Great Escarpment (Fig. 1), where change in climate occurs. Respiratory allergy sufferers in Port Elizabeth and East London rapidly recover and maintain good allergic health in Cradock or Graaff Reinet. Indeed, in these inland towns there are schools in which a proportion of the scholars are from homes at the coast where they cannot live on account of bronchial asthma or nasal allergy.

From a consideration of the cases of perennial respiratory allergy studied at the coastal and inland towns of South Africa it is concluded that climate is the agent responsible for the precipitation of symptoms.

THE CLIMATE FACTOR IN PERENNIAL RESPIRATORY ALLERGY AT THE COAST

Climate is the cumulative weather state over a period of time and is the resultant of air temperature, barometric pressure, rainfall, relative humidity, hours of sunshine and so on. Further, climate is closely related to geographical features such as altitude and proximity to the sea or to mountain ranges.

The complexity of climate makes an analysis of its components necessary in order to identify those which may be of significance in the allergic health of man. The metereological data available have been studied from many points of view, and large numbers of charts based on such data were drawn in the attempt to define critical differences between inland and coastal climates.

Experience with patients in all parts of South Africa had lead to the opinion that the *combination* of high temperature and high atmospheric moisture-content was probably significant. This led to the drawing of further charts incorporating figures of absolute and specific humidity, rainfall, wet-and-dry-bulb readings with those of temperature. However, sufficiently characteristic climate patterns did not emerge.

The possibility was considered that a constantly warm and moisture-laden atmosphere could maintain a congested state of the nasal and bronchial mucous membranes and so increase susceptibility to the allergenic attack, or could perhaps render more highly allergenic otherwise harmless substances. It was thus desirable that a chart should indicate whether a person was living in warm, moist surroundings for all or part of the time, and whether he enjoyed periods of freedom from such conditions during part of the day or part of the year or both. Charts were therefore drawn showing average morning and afternoon temperatures and relative-humidity percentages for each month of the year. This type of chart seemed to fulfill the requirements and was adopted for the present study.

The structure of a climate pattern is shown in detail in Fig. 4, in which Kroonstad, a Highveld town in the Orange Free State, is represented. It will be observed that considerable metereological information is given in a single chart, viz. the average monthly temperature and relative humidity at 8 a.m. and at 2 p.m. throughout the year. A close study of the details is however not essential for a general understanding of the climatic conditions in any particular town. The resulting 'pattern' is obvious and striking. Thus, from a mere glance at the chart it is learned that in Kroonstad there is a wide

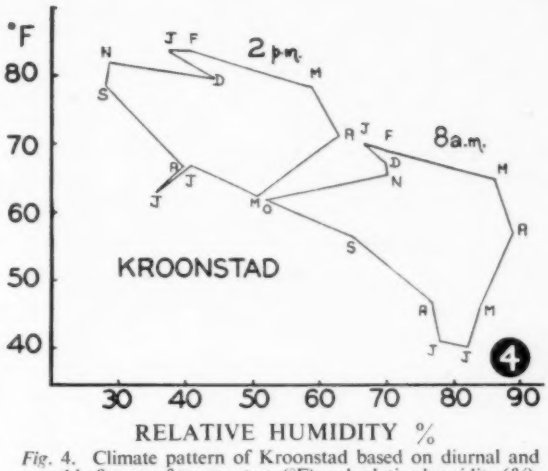

Fig. 4. Climate pattern of Kroonstad based on diurnal and monthly figures of temperature (°F) and relative humidity (%).

diurnal and annual range of temperature and relative humidity.

The climate pattern of a South African coastal town is quite different from that of an inland town and is characterized by a comparatively narrow range of temperature and of relative humidity through the day and during the year. The temperature range is approximately between 55° to 80°F and the corresponding relative humidity between 60 and 80%. Even a cursory look at the metereological chart of a coastal town reveals the typical 'compressed' climate pattern almost entirely confined to the upper right-hand quadrant, indicating high temperature and high relative humidity with little variation during the day and throughout the year.

In Fig. 5 the climate patterns of 10 inland and 5 coastal towns of the Union of South Africa are shown together for comparison. The coastal towns are represented in the vertical column on the right of the figure. The contrast is to be noted of the climate patterns of the inland towns with their wide and the coastal towns with their narrow ranges of temperature and relative humidity. Some of the inland towns in the Highveld (vertical column on the left) show a very marked change in the month-to-month temperature and relative humidity both for day or night whereas in other towns not on the Highveld (central vertical column) variations from month to month are not so great but the day-night variations are nevertheless pronounced. A study on these lines of the climate pattern of any town in South Africa should help the physician to decide whether his allergic patient whose symptoms are considered to be precipitated by climate may safely visit that town or should avoid it.

It is not sufficient to invoke altitude alone in explanation of the observed clinical differences in low-altitude coastal and high-altitude inland areas, because it is probable that perennial respiratory allergy of climatic causation also occurs in certain inland districts of South Africa. The humidity factor inland appears to

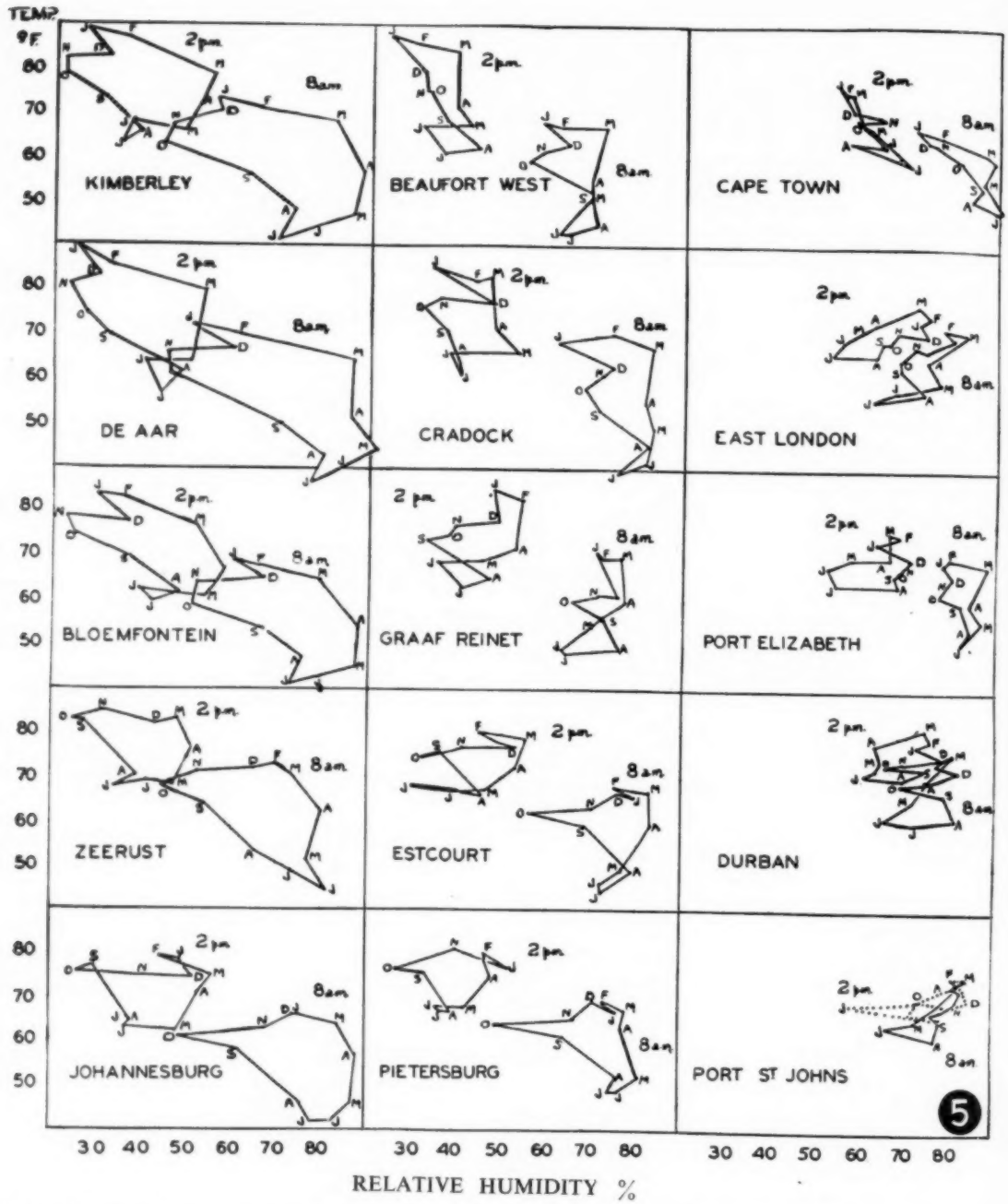

Fig. 5. The climate patterns of inland and coastal (*vertical column on the right*) towns of South Africa based on diurnal and monthly figures of temperature (°F) and relative humidity (%).

be associated with mists. The subject is being investigated and climate patterns of towns in the 'mist-belts' are in preparation.

It has been mentioned already that in the coastal towns of the Union, 'good' and 'bad' suburbs for asthmatic and nasal allergy sufferers are recognized. This subject is being studied, but it is not easy scientifically to confirm these clinical impressions because the relevant metereological data are lacking and because other possible explanations so far considered have not proved satisfactory.

THE CLINICAL APPROACH TO CASES OF PERENNIAL RESPIRATORY ALLERGY OF SUSPECTED CLIMATE ETIOLOGY

The problem of perennial respiratory allergy in South Africa initiated or aggravated by climate is not an easy one for the physician or his patient. The present diverse views on the etiology of coastal respiratory allergy result in frustrating attempts at control with drugs or by desensitization procedures on the basis of skin-test reactions. The knowledge that climate factors exist and may operate is a step forward. A clearly-defined approach to the patient becomes possible. The physician is on firmer ground with regard to the difficulties to be faced and realistically dealt with.

Patients who maintain reasonably good allergic health inland and tend to breakdown at the coast are well advised to avoid the coast. Even short visits there may precipitate allergic exacerbations which may continue for weeks or even months (Figs. 2 and 3). Coastal sufferers, especially children, should unhesitatingly be advised, where possible, to leave the coast and reside inland. The question of the optimum period of absence from the coast is as yet undetermined and must of course vary with different patients. There is some evidence however, that an absence of at least 2 years is necessary to reduce the liability to allergic breakdown. In any event in an inland area the patient will no longer be subjected to allergic depletion and will be given the opportunity of normal physiological progress. It is likely that the longer he remains free from the physical and emotional stresses of respiratory allergy the better will he be able to withstand the hazards of his return to the coast should this become necessary.

The approach to the allergy patient suggested above may prove a drastic one in some cases but until more is learned about simpler prophylactic and therapeutic measures avoidance of the coast, especially by young patients, must be insisted upon. Such a recommendation, however, which may involve considerable domestic, social and financial disturbance, should be made with the greatest circumspection after reasonable therapeutic trials and after thorough clinical and laboratory investigations have failed to reveal exogenous or endogenous factors in explanation of the continuance of symptoms.

SUMMARY

Seasonal respiratory allergy in South Africa is almost entirely a grass pollinosis occurring in the summer from October to March. Perennial respiratory allergy is due to exogenous and endogenous factors similar to those found elsewhere.

The incidence and the severity of the perennial type of respiratory allergy, including bronchial asthma and nasal allergy, are relatively great in the coastal areas of South Africa, more especially on the East Coast.

Numerous cases seen in Johannesburg and in various coastal towns are described and these demonstrate the fact that persons maintaining good allergic health inland may break down at the coast, and that symptoms in coastal sufferers are ameliorated or cease when they move inland.

Climate is regarded as the important exciting agent in perennial respiratory allergy at the coast.

The *combination* of high atmospheric temperature and high relative-humidity in constantly narrow range throughout the day and during the year appears to be the significant climate factor.

Marked and characteristic differences in climate patterns between inland and coastal areas are revealed in a series of charts based on these temperature and relative-humidity factors.

Climate patterns of the type shown may guide the physician in deciding which part of the country his patient should avoid or need not avoid.

The appreciation of the importance and significance of climate in perennial respiratory allergy should lead to a more clearly defined approach to the control of sufferers.

Patients who remain allergically well inland should avoid the coastal areas and sufferers at the coast should if possible live inland. In this connection the importance of a thorough preliminary investigation from the clinical and laboratory point of view cannot be too greatly stressed.

The writer gratefully acknowledges the assistance he derived in his studies of weather and climate in South Africa from conversations with Mr. B. R. Schulze of the Weather Bureau in Pretoria, Dr. W. Schaffer of the University of Cape Town, and Dr. S. P. Jackson of the University of the Witwatersrand. Professor J. H. Wellington of the University of the Witwatersrand kindly supplied relevant geographical information.

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Especial thanks are due to numerous physicians, paediatricians, otorhinolaryngologists and general practitioners in Johannesburg, Cape Town, Port Elizabeth, East London and Durban, who very kindly discussed their problems with the writer and placed at his disposal for clinical and laboratory study many of their patients suffering from allergic respiratory conditions.

The metereological data were obtained from the report [6] of the Weather Bureau of the Union of South Africa, 1950.

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THE ULTIMATE RESIDUAL LESIONS OF ASPHYXIA NEONATORUM

WITH THREE CASES OF PORENCEPHALY

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The aetiology of the degenerative brain conditions of infants and children is as yet not fully understood. Congenital and infective causes, as well as birth injuries, are recognized as important causes, but it is only recently that the role played by anoxia as an aetiological factor is becoming more fully realized. The majority of these brain lesions have been caused experimentally by exposing laboratory animals to anoxia. Furthermore, in many cases of degenerative brain conditions which are diagnosed clinically or at post-mortem examination, a history of asphyxia neonatorum is obtained; in one series of cases,[13] a history of apnoea at birth was obtained in 70% of 500 children with neurological abnormalities. As is well known, anoxia may cause cerebral palsy and, as Evans [6] believes, usually of the athetotic type.

CONDITIONS WHICH MAY BE CAUSED BY ASPHYXIA NEONATORUM

I. Courville [2] divides these results into 4 groups:

1. *Diffuse Loss of Nerve Cells.* In this condition there is, according to Courville, such a diffuse loss of nerve cells that the convolutions of the brain are smaller than normal, but there are no recognizable changes in the internal structure. Experiments [17] were carried out in which the uterine arteries of gravid guinea-pigs were temporarily occluded, and the subsequent progeny showed either localized or diffuse atrophy of the brain cortex as the result of generalized loss of nerve cells. The corpus striatum is also extremely sensitive to anoxia and Courville is of the opinion that this is an important cause of juvenile Parkinsonism and congenital choreo-athetosis.

2. *Cortical Sclerosis, generalized or localized.* In this condition atrophy and sclerosis of the convolutions result and this may be generalized or localized. Courville[2] quotes a case in which Meyer found atrophic nodes of the cortex at the post-mortem examination of a patient who 16 years previously had suffered from carbon-monoxide poisoning—the obvious cause of the anoxia. He supposes that the formation of scar tissue in the cortex, following exposure to anoxia, may eventually lead to epileptiform attacks. This type of lesion has also been found in cases of spastic paralysis of children.

3. *Disorders of Myelination.* Encephalitis periaxialis diffusa (Schilder's disease) was previously considered to be of inflammatory origin, but this idea has been largely abandoned. In 1926 [8] its similarity to demyelination, which follows on carbon-monoxide poisoning, was noticed. Courville considers asphyxia neonatorum or intra-uterine anoxia to be the most likely cause both of the infantile and the subacute type of Schilder's disease in children.

4. *Cyst-formation and Porencephaly.* In early life 2 other cerebral lesions occur, viz. chronic infantile cystic degeneration and porencephaly. The former condition was at first considered to be of inflammatory origin, but its early association with birth injuries and subdural haematomas pointed to a traumatic origin. This was confirmed by the experimental production of cystic degeneration of the brain as a result of cerebral anoxia caused by inhalation of carbon-monoxide and nitrous oxide.

Perhaps the most widely accepted definition of porencephaly is that of Le Count and Semerak,[18] who describe it as 'a defect communicating with the ventricles or separated from them by a thin layer of brain tissue, and covered on the outside by the arachnoid'. The different aetiological factors causing porencephaly may be classified as developmental, traumatic, vascular and inflammatory. Traumatic and vascular factors are closely interrelated. Vascular accidents such as haemorrhage, embolism and thrombosis may lead to cerebral anoxia, with porencephaly as the result. This has been observed experimentally as well as clinically. Inflammatory lesions such as tuberculosis, syphilis and meningo-encephalitis have been described as underlying causes of porencephaly. 'The very multiplicity of possibilities in many instances makes it difficult to establish the underlying cause.' [18] In a series of 29 cases of Pendergrass and Perryman [18] the aetiology was not definitely determined in 17 cases. Five of their cases were apparently caused by intra-uterine or birth injuries, 3 by trauma in later life and 2 by encephalitis. It is clear that porencephaly may follow neonatal asphyxia, and its recognition is important in determining prognosis and in differentiation from mass-producing or constricting lesions involving the brain. Of their 29 cases 26 sought medical attention because of epileptic attacks, while the others complained of headache, poor memory, irritability and mental retardation. In 2 of our cases the chief complaints were excessive developmental and mental retardation, both with complete blindness and one with repeated convulsions. The 3rd case presented with extreme spasticity.

A definite diagnosis of porencephaly can be made only by the use of pneumo-encephalography, and porencephaly may still be demonstrated by films taken 24 hours after the injection of air into the lumbar subarachnoid space.

II. *Other Effects on the Central Nervous System*

1. *Intelligence.* It is difficult to assess the effect of asphyxia neonatorum on the intelligence. It can be done by obtaining the birth history of the mentally retarded children. Campbell *et al.*[1] point out that a history taken from a mother after the passing of several years may be inaccurate or may be unduly modified in order to give a reason for her child's mental condition. The Darke's studies [4] show that children may well have subnormal intelligence as a result of asphyxia neonatorum. Other

investigators [1, 16] were not able to elicit proof of a greater occurrence of lowered intelligence in these cases as compared with normal controls. The answer will only be found after large series with adequate follow-up investigations have been studied.

2. *Behaviour Disorders.* It is well known that changes in behaviour can occur after the administration of anaesthetics, especially nitrous oxide. This is attributed to anoxia. Fletcher [7] described 27 cases following nitrous oxide anaesthesia. The disturbances may occur early or years afterwards; they may improve within months or over a number of years or may become progressively worse. Amongst others, he noticed the following symptoms: mental confusion, poor concentration and memory, behaviour disorders, apathy, hysteria, restlessness and headache. He believes that the sensitive cortical cells are damaged by the anoxia. The condition may improve in time, but where the damage is extensive the changes may be irreversible. He suggests that repeated administration of anaesthetics to children, and asphyxia neonatorum, may result in behaviour disturbances.

Preston [11] described 132 cases of behaviour abnormalities in children with a history of anoxia at birth. He distinguishes three groups: (*a*) with hyperactive behaviour—37%, (*b*) with apathetic behaviour—34%, and (c) with epileptic behaviour—29%. The intelligence of 35 of the 132 cases was subnormal and the rest normal, but all were problem children.

Rosenfeld and Bradley [12] perused the records of 673 children with abnormal behaviour. In a large percentage a hisotry of apnoea at birth or of pertussis before the age of 3 years was found; the only factor which these two conditions have in common is anoxia.

3. *Epilepsy.* Epilepsy as a late result of anoxia is well known. Preston [11] also mentioned this in his series of cases. Courville [2] believes that anoxia causes cortical sclerosis resulting in epileptic seizures. Nielsen [9] is of the opinion that anoxia is responsible for some cases of idiopathic epilepsy. Instances of epilepsy have been reported in pilots who have flown at great heights with inadequate oxygen supplies.

4. *Hemiplegia.* At the post-mortem examination in a case of a woman aged 80 years with hemiplegia since birth a cyst was discovered in the internal capsule together with widespread degenerative lesions of the brain.[3] These findings indicated anoxia as the causal factor.

5. *Electro-encephalographic changes.* In ECG investigations carried out in Sweden [5] on 61 children who had had asphyxia neonatorum 4 to 7 years previously, 24 (40%) showed abnormal rhythm and low-frequency waves. It is stated that only 5% of normal children shows an abnormal encephalogram, which makes the high percentage in the series significant.

As an example of the late results of antenatal and neonatal asphyxia, three cases are presented where porencephaly was found as the ultimate residual lesion.

CASE 1

A Bantu boy 18 months old was admitted on 8 March 1954 to Professor Davel's paediatric wards. The patient's neck and arms had been flaccid since birth. He was still unable to lift his head or sit or crawl and was incapable of any speech. From birth feeds had been taken with difficulty and growth was retarded.

Birth History. He was the firstborn baby, full term. The confinement at home, without professional aid, was difficult and protracted, labour lasting 2 days. Breathing and crying were absent immediately after birth, cyanosis was extreme and resuscitation unsuccessful, the infant being presumed dead. Two hours later, however, it was noticed that the baby was alive.

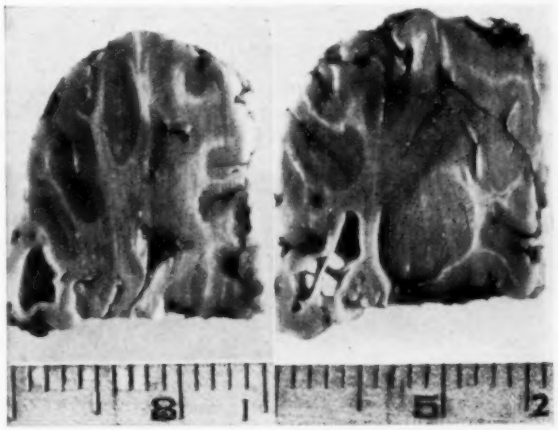
On Examination. The patient was emaciated and underdeveloped; the head was retracted and mouth gaping. Weight 13 lb. 1 oz.; head circumference 17 inches; anterior fontanelle was almost closed.

Central Nervous System. The patient was obviously mentally and physically retarded. The cranial nerves were normal except for an internal strabismus of the right eye. The neck was flaccid, with hypotonia of the arms and decreased tendon reflexes. There was hypertonia of the lower limbs with slight adductor spasm.

No further abnormalities were found in any of the other systems.

Special Investigations. Roentgen photos of the skull were normal except for microcephaly. C.S.F. showed no abnormalities. The blood picture was normal. Before any further investigations could be carried out, the child suddenly developed hyperpyrexia and died.

Post-Mortem Examination. After removal and fixation of the brain, sections showed 2 subcortical cysts in the left frontal region (Figs. 1 and 2). Histological examination of the cortex and basal ganglia (by Prof. J. Barnetson) revealed no obvious changes. No further abnormalities excepting a fatty infiltration of the liver were found.



Figs. 1 *and* 2. Case 1. Sections of the left hemisphere show the 2 subcortical cysts with no communication with the ventricular system. An encephalogram would have been negative in this case.

CASE 2

A European baby-girl 3 months old was admitted to the paediatric wards for the first time on 23 September 1953, suffering from gastro-enteritis. Since then she has been admitted on 3 other occasions with the same complaint. On the 4th admission (2 February 1954) at the age of 7 months, it was noticed for the first time that she was unable to hold her head erect, was not able to sit upright and could not observe objects. Examination revealed a head circumference of 16 inches and commencing bilateral optic atrophy. Roentgen photos of the skull showed no abnormalities but, after 25 c.c. of air had been introduced by the lumbar thecal route, a shadow was demonstrated above and lateral to the right lateral ventricle (Fig. 3). On the last admission (15 September 1954) at the age of 15 months, the head circumference was 16½ inches and complete primary optic atrophy was present. The developmental age at this stage is that of a baby of 2 months. She is not able to keep her head erect or to sit up. The Moro reflex is still present. Special investigations of the blood and C.S.F. gave normal results; the Kolmer reaction was negative.

Antenatal and Birth History. About 2 weeks after the expected menstrual period the mother had a slight vaginal haemorrhage lasting 1 day. A week later she had another haemorrhage lasting 2 days followed 6 weeks later by a severe haemorrhage for which she was admitted to hospital and treated for a threatened abortion. After this everything went well until the 7th month, when she again had a severe haemorrhage and this time was admitted as a suspected placenta praevia. The bleeding subsided and she was sent home again until labour commenced at full term. It was very difficult and lasted several hours. According to the mother, no instruments were used, but the head was manually rotated and a large episiotomy done. The baby was very blue; the blueness lasted for 3 days and gradually disappeared. The birth weight was 5½ pounds.

CASE 3

A 13-month-old Bantu baby-boy was admitted with gastro-enteritis; further history elicited mental and developmental retardation with suspected blindness. A month before admission his left leg became spastic.

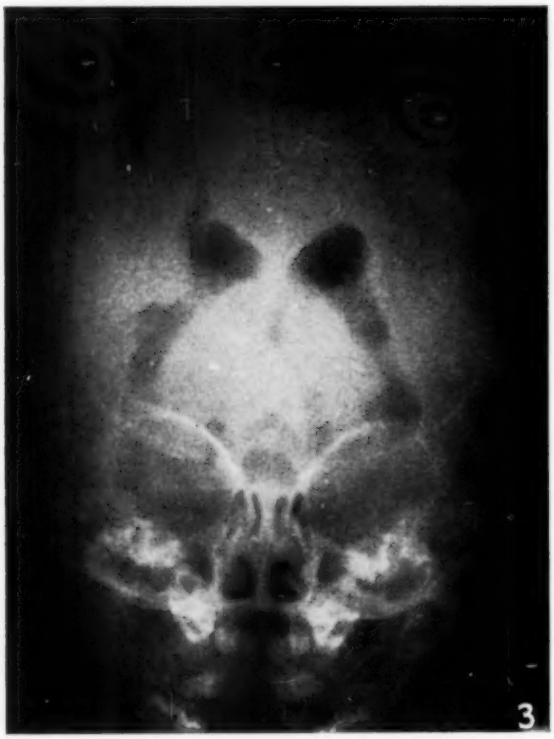
Pregnancy history revealed no haemorrhages, illnesses or virus infections. Labour itself was considered to be normal, but this history is usually unreliable with Bantu people.

On examination, the child showed slight dehydration. He was blind and could neither keep his head erect or sit up. The Moro reflex was still present and the left leg was slightly spastic. His head circumference was 16 inches and he was completely blind, with complete optic atrophy. Special examinations of the blood and C.S.F. were normal, with negative Kolmer tests. A pneumo-encephalogram revealed porencephaly (Fig. 4).

Comment. In the first case prolonged asphyxia neonatorum obviously caused severe anoxia with the results stated. In case 2 there was an additional history of antenatal haemorrhage and one wonders what influence the small bleedings in early pregnancy could have had. Case 3 gave no history of any antenatal or neonatal asphyxia, but the baby revealed the same condition as the other two. Pneumo-encephalography showed the porencephaly to be more widespread than in the other two cases.

Discussion: It is now believed that asphyxia neonatorum and antenatal anoxia may have serious residual effects, not only as an immediate, but also as a late complication. Parturition [10] is the most dangerous experience to which the individual is normally subjected and one of the greatest dangers is anoxia. For this reason there are different mechanisms [14, 15] for the protection of the foetus and neonatus against this danger. The most important factor is probably the greater affinity of foetal haemoglobin for oxygen in comparison to that of the adult. Furthermore, the metabolic rate of the brain of the newly-born is low and anaerobic conversion of blood-sugar occurs. Should the apnoea be of long duration, it may give rise to irreparable damage of the sensitive nerve cells with varied resultant clinical entities such as porencephaly, Schilder's disease, cerebral palsy, epilepsy, mental retardation, and others.

In case 1, where anoxia was of long duration, the clinical findings were those of cerebral palsy and this, together with the post-mortem findings of porencephaly,



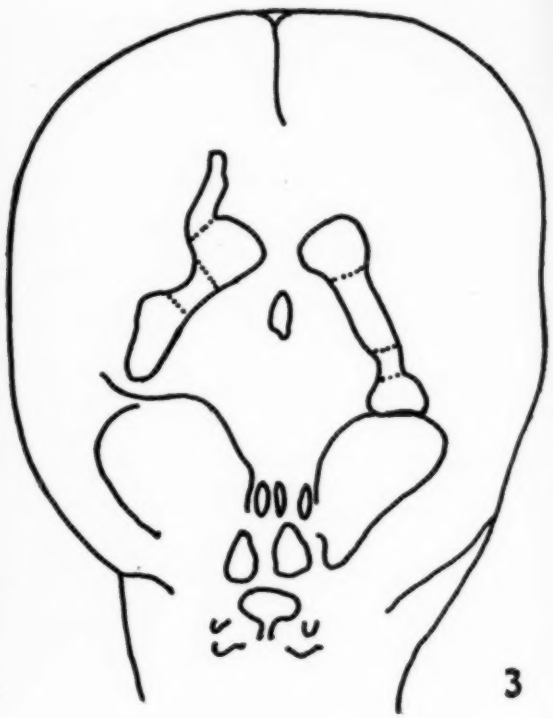
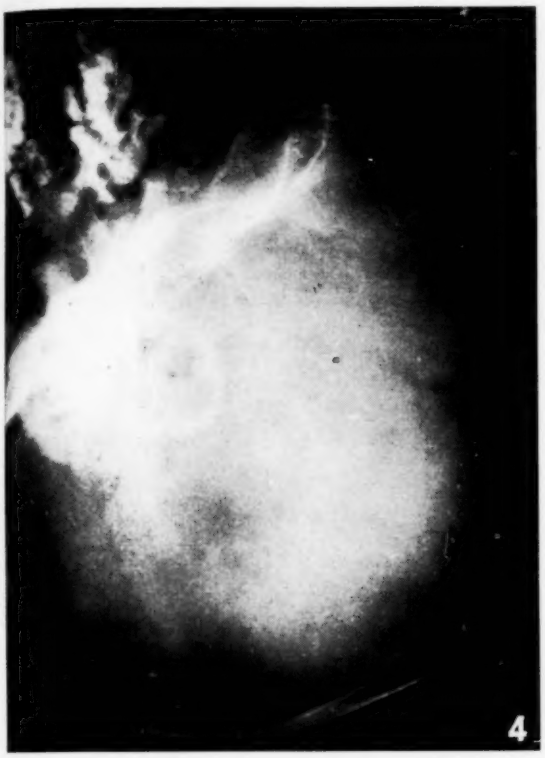


Fig. 3. Case 2. Encephalogram illustrates porencephaly in the left hemisphere communicating with the anterior cornu of the right lateral ventricle. Also diagram of same.



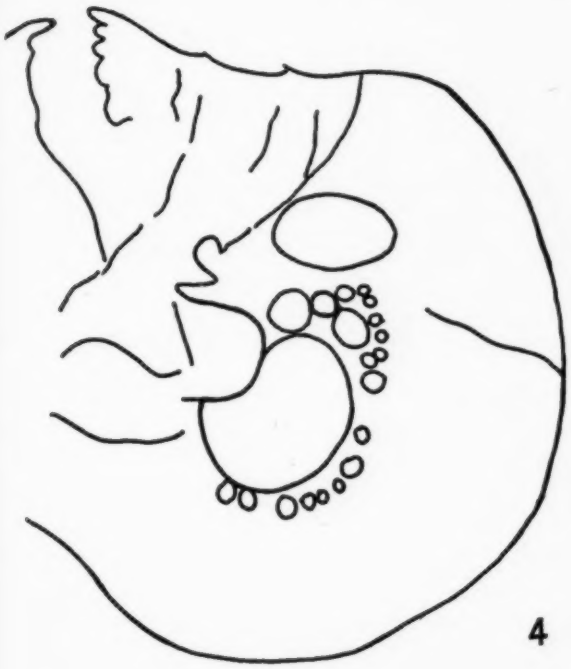


Fig. 4. Case 3. Encephalogram shows a cluster of 'daughter' cysts on the wall of a large 'mother' cyst; this is very rarely seen. Also diagram of same.

are typical ultimate residual lesions of anoxia. There is adequate evidence that anoxia plays an important role in the aetiology of a large group of apparently unrelated degenerative conditions of the central nervous system. Two changes are thought to occur: Firstly, where damage to the nerve cells is minimal, there is every possibility, according to Fletcher,[7] of improvement taking place with the passing of months or years. This was proved by his observations on patients with mental disorders following repeated general anaesthesia. Preston's observations on children with behaviour disorders as a result of neonatal anoxia substantiate this view.[11] Secondly, where the damage is extensive, degenerative brain lesions develop.

Courville supposes that anoxia produces minimal functional changes in the nerve cells which shorten their life-span. The cells, after functioning normally for several years, start deteriorating. Thus symptoms may only occur in later childhood or even during adult life.

SUMMARY

It is now accepted that anoxia (antenatal, natal and postnatal) is a most important single factor in the development of degenerative lesions of the central nervous system in children and probably in adults as well. Conditions such as cerebral palsy, Schilder's disease, porencephaly, epilepsy, juvenile Parkinsonism, etc., may be caused in this manner. Anoxia may likewise be responsible for some behaviour disorders and lowered intelligence in children. Three cases with porencephaly are described, 2 of them following prolonged post-natal and antenatal anoxia. Similar changes caused by asphyxia neonatorum and prenatal anoxia are described in the literature reviewed.

OPSOMMING

Dit word aanvaar dat asphyxia neonatorum, asook prenatale anoksie 'n belangrike rol speel in die etiologie van degeneratiewe breinsiektes van babas, kinders en miskien ook van volwassenes. Toestande soos serebrale verlamming, Schilder se siekte, porenkefalie, epilepsie, Parkinsonisme van jeugdiges ens., mag hierdeur veroorsaak word. Dit mag ook verantwoordelik wees vir gedragsafwykings en lae intelligensie by kinders. Drie gevalle van porenkefalie bewys met pneumo-enkefalografiese ondersoeke en lykskouing word beskryf. In 2 gevalle is daar 'n duidelike geskiedenis van prenatale en postnatale anoksie. Die kliniese verskynsels van hierdie drie gevalle het bestaan uit geestelike en liggaam-

like agterlikheid, mikrokefalie, optiese atrofie en serebrale verlamming.

We express our appreciation to Professor J. G. A. Davel for his guidance and assistance with these cases.

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CERTAIN ASPECTS OF TRANSFUSION UNDER PRESSURE

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Increased use is being made of positive pressure for blood replacement by the intravenous route. Not only is it a suitable means of transfusion where blood loss has resulted in venous collapse or spasm, but it is proving useful for rapid repletion of blood volume after severe haemorrhage during thoracic, and in particular cardiac and vascular, surgery. Coupled with this is the development of intra-arterial transfusion as an improved means of resuscitation.[1-3] A sure and safe method of avoiding air embolism is of prime importance. This has prompted the description of a simple piece of apparatus which is coming into common use for positive-pressure transfusion.

THE DANGER OF AIR EMBOLISM

The existing type of drip feed used in transfusion apparatus lends itself to the danger of rapidly fatal air embolism when positive pressure is applied to the transfusion bottle.[4, 5]

The commonest way in which air embolism has occurred is as a result of air being forced into the tubing after the level in the transfusion bottle has reached the level of the outlet tube. The precaution of keeping a continuous watch on the fluid level is not enough, for there is another less obvious means by which air embolism may result, which is demonstrated by the following case report.

Case Report. B.W. aged 54 years. Left pneumonectomy for extensive bronchiectasis. Because of considerable pleural adhesions, operating time was prolonged and blood loss necessitated the transfusion of 5 pints of blood, aided by positive pressure from a double bellows. A constant watch was kept on the transfusion bottle. When about 200 ml. of blood remained in the 5th bottle, it was noticed that a mixture of blood and air was entering the drip feed, despite the fact that the pressure in the bottle was restored to atmospheric level by disconnecting the bellows. The blood level in the dripper dropped completely. The tubing was immediately removed where it joined the intravenous needle, and a new transfusion set up. The patient suffered no untoward reaction.

On examining the apparatus it was found that, when it was allowed to flow, a short column of pure blood was followed by a mixture of air and blood. This mixture continued to flow despite the fact that the bottle was about half full and although no positive pressure was applied. When the 'wick' filter which had been continuously used for the previous 4 pints of blood and half of the 5th pint was examined, it was found that the mesh had been partially blocked with sediment. This caused the blood outside the 'wick' filter to dam up and the blood within the confines of the filter drained away and was replaced by air.

Dolten *et al*,[6] in 1945, reported on a similar occurrence, with a fatal result, due to this type of air embolism caused by a partially blocked filter. They pointed out that fatalities, in fact due to this type of air embolism, may in the past have been mistaken for status lymphaticus, acute cardiac failure, shock, and the like.

It must be stressed that this progressive blockage of the filter by sediment, with its attendant danger of air embolism, readily occurs when gravity alone is employed to transfuse blood. The danger may be avoided by regular renewal of the filter in the course of prolonged transfusion.

Apparatus

Fig. 1 illustrates the conventional transfusion apparatus designed so that positive pressure may be safely applied.

The glass drip-chamber is replaced by one which contains a float. The lower end of the float and the lower end of the drip-chamber are so ground as to create an airtight fitting when they come into contact with each other. The tubing and drip-chamber are filled in the same way as the ordinary apparatus. Details of this 'safety dripper' are illustrated in Fig. 3. The side tube allows for pressure equalization when the clip is opened. This permits the level of the float to be set at any desired height above its seating.

Fig. 2 indicates what occurs when the transfusion bottle empties. Air forced into the drip chamber causes the fluid level to drop. The float then sinks onto its seating, sealing the apparatus off from any further flow, and thus air embolism is prevented.

A screw cap, the top of which has been holed to allow passage of the glass inlet and outlet tubing is shown in Fig. 3. Figs. 1 and 2 show the purpose of this cap,

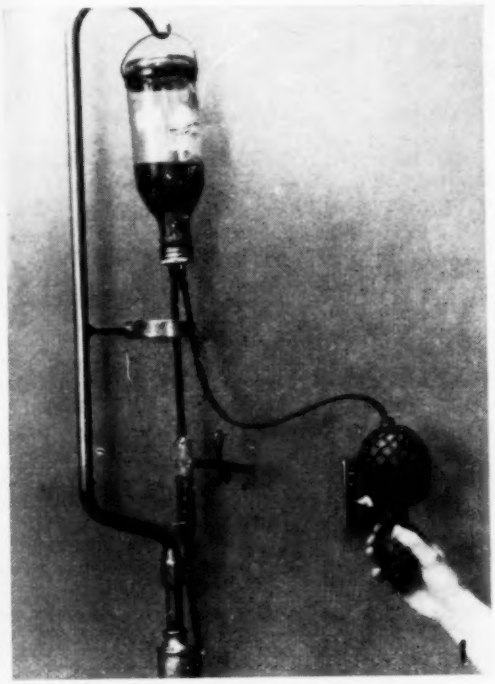

Fig. 1

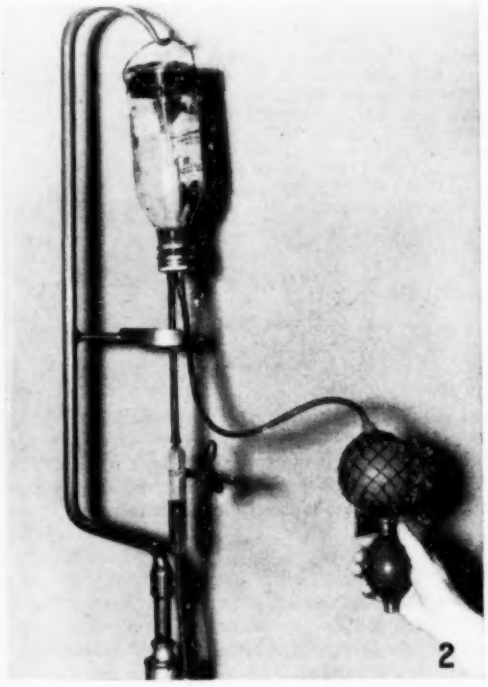

Fig. 2

which is to hold the rubber cork firmly in the rim of the bottle so that any excess of pressure will not blow cork, tubing and filter out of the transfusion bottle. This cap must be deeper than the usual screw cap, so as to accommodate the protruding part of the rubber cork and at the same time take a good grip of the rim of the bottle.

Positive pressure is obtained by the use of a double hand-bellows (Figs. 1 and 2), which allows an even and adequate head of pressure to be maintained. No other method, such as the employment of an oxygen cylinder, is advisable.

Fig. 3. Safety dripper and modified cap.

For intra-arterial transfusion all metal and rubber joints should be secured by wire. Also an 'aneroid' type of pressure gauge may be coupled (by a 'Y' tube) to the double bellows to indicate the pressure in the transfusion bottle.[7]

Testing of the Safety Dripper. In view of the less-known cause of air embolism (due to a partially clogged filter) we tested the reaction of the float mechanism when a mixture of blood and air entered the upper end of the drip chamber. The entry of such a mixture caused the float to fall with the receding fluid level in the drip-chamber. Any further flow was prevented as effectively as when air *alone* entered from the drained transfusion bottle.

In a further experiment carbon dioxide dissolved in saline at a pressure of 80 mm. of mercury was run through the apparatus. This solution immediately on reaching the drip-chamber again caused the float to

prevent any flow. Here the gas is released from solution and depresses the fluid level and the float with it.

OTHER ASPECTS

Rate of Blood Flow.[1-3] Jones [8] has recently emphasized that the size of the bore of the transfusion needle is the main factor limiting the volume transfused where gravity is the force employed. Where positive pressure is used a large-bore needle is just as important.

Instead of a large-bore needle, polythene tubing of 1·5 mm. bore may be used, in the following manner: The tubing is introduced by threading it through a suitable cannula. It is then passed up a few inches along the length of the vein, allowing it to reach a vein of wider lumen. The advantages are obvious. Little splinting of the limb is required and there is minimal risk of dislodgement from the vessel. The use of polythene tubing is also most satisfactory where large quantities of blood or packed cells are to be administered over a long period.

Venous spasm or collapse is commonly encountered as a result of massive haemorrhage. Here, however wide the bore of the needle, positive pressure will be essential to assure adequate transfusion, if irreversible shock is to be avoided. Flow rates of 75 to 350 ml. per minute have been safely maintained under positive pressure *via* the arterial or venous route. Experience, however, has shown that for maximum safety the total volume replaced in this way should not be more than 70% of the estimated volume lost through haemorrhage. Where jugular tracings have been taken in these cases, they have shown no undue increase in venous pressure.

Where surgical procedures are undertaken which involve sudden grave loss of blood, a reasonable estimate of the quantity lost may be made by weighing all swabs before and after use. All clots removed from the wound and all blood taken up by the suction apparatus must of course also be weighed. Thus replacement volume for volume is much more accurately and beneficially achieved.

Intra-arterial Transfusion. Those contemplating the use of intra-arterial transfusion must be fully aware of the possible complications (apart from air embolism) which beset this newer method of resuscitation. Complications include the effect on the myocardium of the excessive plasma potassium in stored blood. Then there is the possibility of small blood-cell emboli reaching the arterial system. There is also the risk of ischaemia of the member distal to the site of arterial cannulation.

Seeley and Nelson [2] and Melrose and Wilson [3] have exhaustively discussed this subject; Devitt and Wigderow [9] report on a successful case of intra-aortic transfusion. These reports are well worth careful study.

SUMMARY

Attention is drawn to the danger of partially clogged blood-transfusion filters which may allow a mixture of blood and air to enter the patient's circulation and cause fatal air embolism.

A description is given of a simple apparatus for safe positive-pressure transfusion. The apparatus was tested under different conditions to prove its efficacy.

Means of achieving a satisfactory rate of flow, and the quantities transfused under positive pressure, are discussed.

A warning is given of the possible dangers of intra-arterial transfusion. These may be avoided if the literature available on the subject is studied.

Thanks are due to Dr. George Edwards, Head, Department of Anaesthesia, St. George's Hospital, for permission to publish the case report.

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FUNGAL DISEASES IN SOUTH AFRICA

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In recent years there has been a world-wide increase of interest in medical mycology. Innumerable articles have been written and several text-books have been published. In some of them the information on the incidence of fungal diseases in South Africa is erroneous or misleading. For example, Conant *et al.*[1] do not include this country in the geographical distribution of Benign Histoplasmosis. They report Maduromycosis in Africa but not specifically in South Africa. Langeron [2] has omitted South Africa in the distribution of Torulosis and states that 3 cases of Rhinosporidiosis have been reported from this country.

No fault attaches to the authors. The reason is that after the first report of certain diseases it was thought that no useful purpose would be served by publishing subsequent cases. However, it now appears advisable to publish the incidence of various mycoses in South Africa.

The figures given below are based entirely on the specimens received at this Institute during the past 8 years. This laboratory serves a limited area and the number of specimens handled by other laboratories is unknown to the author. Moreover, the large majority of cases of the dermatomycoses are diagnosed clinically,

and only where the diagnosis is in doubt are specimens sent to the laboratory. Another fact to be borne in mind is that those areas of South Africa which are set aside as Native (Bantu) Reserves possess few medical or laboratory facilities. In one such area it is known on clinical grounds that Favus is extremely common but this is not apparent from an analysis of the figures based on specimens received at the laboratory. The following figures, therefore, do not indicate the total incidence of mycoses in South Africa, nor do they reflect accurately the relative incidence of different mycoses.

DERMATOMYCOSES. All types of dermatomycosis occur in S. Africa but no figures are available on their relative incidence. Any deduction from our specimens would be misleading, since most cases are diagnosed on clinical grounds alone. It is of interest, however, to note the relative frequency of the dermatophytes

TABLE I. DISTRIBUTION OF DERMATOPHYTES ISOLATED AT THE INSTITUTE DURING THE PAST 8 YEARS

	Number	*Percentage*
Microsporum:		
canis	161	73
gypseum	2	1
audouini	5	4·5
Epidermophyton:		
floccosum	11	5
Trichophyton:		
mentagrophytes	16	7
rubrum	1	0·5
violaceum	9	4
schoenleini	9	4
concentricum	1	0·5
discoides	1	0·5
Total	216	100

TABLE II. TOTAL NUMBER OF CASES OF VARIOUS FUNGAL DISEASES DIAGNOSED AT THE INSTITUTE DURING THE PAST 8 YEARS

Otomycosis	19
Sporotrichosis	34
Chromoblastomycosis	41
Maduromycosis	22
Actinomycosis and Nocardiosis	96
Rhinosporidiosis	12
Cryptococcosis	18
Histoplasmosis	2
Pulmonary Aspergillosis	2

encountered on the Witwatersrand. In 221 positive cultures the distribution was as follows: *Microsporum canis* 73%, *Trichophyton mentagrophytes* 7%, *Epidermophyton floccosum* 5%, *Microsporum audouini* 4·5%, *Trichophyton violaceum* 4%, *Trichophyton* (*Achorion*) *schoenleini* 4%, *Microsporum gypseum* 1%, *Trichophyton rubrum* 0·5%, *Trichophyton concentricum* 0·5% and *Trichophyton discoides* 0·5%. The results are summarized in Table I.

A noteworthy feature is the infrequency of *Microsporum audouini* and *Trichophyton rubrum* as compared with their incidence in Great Britain.

Tinea versicolor and Erythrasma are common and Trichomycosis axillaris is occasionally encountered but neither Piedra nor Tinea nigra palmaris has been seen by the author.

OTOMYCOSIS is fairly common (19 cases) and the fungi most frequently found are species of *Aspergillus*, particularly *A. niger*.

SPOROTRICHOSIS. During 1942, 1943 and 1944 an epidemic of over 2,000 cases of Sporotrichosis occurred among gold miners of the Witwatersrand.[3] As a result of chemical treatment of the timber, the disease has practically been eradicated from the mines but sporadic cases still occur, and during the past 8 years 34 cases have been diagnosed at this Institute.

CHROMOBLASTOMYCOSIS. Since Simson [4] reported the first cases of chromoblastomycosis in this country, a further 41 have been encountered. The majority of cases occurred in Bantu subjects and diagnosis was established histologically. In 6 cases from which tissue was submitted for culture the *Hormodendrum pedrosoi* was isolated. In all 6 strains, only hormodendrum and acrotheca types of sporulation were found. No phialophora cups were seen in any of the strains in spite of repeated search on a large variety of media in which various carbohydrates, trace elements and vitamins were incorporated.

MADUROMYCOSIS. 22 cases were diagnosed on histological section. Of these the majority occurred on the foot, 2 on the hand and 1 on the scalp. In a few instances the site was not specified. The *Madurella* was recovered from 3 cases (2 hand, 1 foot) and the *Monosporium apiospermum* from 1 (foot). The remaining cases were not cultured.

ACTINOMYCOSIS AND NOCARDIOSIS. These are the most common of the deeper mycoses; 96 cases were encountered, over 75% of which were in Bantu subjects. In 80 cases in which the anatomical site was known, the distribution was as follows: Skin and subcutaneous tissue 79%, lungs 14%, lymph nodes 4%, joints, liver and ovary 1% each. The diagnosis was established by cultural examination of pus or biopsy specimens in 27 instances. In 57% of these cases the organism responsible was *Actinomyces israeli*, in 35% it was *Nocardia asteroides*, in 4% *N. gypsoides* and in 4% *N. madurae*.

RHINOSPORIDIOSIS. A total of 12 cases was diagnosed. Of these 9 involved the eye (7 Bantu and 2 European) and 3 the nose (1 Bantu and 2 Europeans).

TORULOSIS (CRYPTOCOCCOSIS). In all, 18 cases were diagnosed. Of these 17 presented with meningitis, of which one also had multiple subcutaneous lesions, and one suffered from a lung abscess.

MONILIASIS (CANDIDIASIS). Amongst the conditions caused by this organism, thrush, glossitis, perlèche, erosio interdigitalis blastomycetica, intertrigo, paronychia and vaginitis are frequently encountered. No cases of conjunctivitis, meningitis * or endocarditis due to this organism have been observed.

The incidence of bronchomoniliasis is, as expected, extremely difficult to establish. Although *Candida albicans* is the most common fungus isolated from sputa it was considered in only 2 cases that a diagnosis of bronchomoniliasis was justified.

BLACK HAIRY TONGUE. This condition is fairly

* For a case of cerebrospinal moniliasis at Cape Town, see Emdin, W. and Finlayson, M. H. (1954): S. Afr. Med. J., **28**, 868.

common. Several cases were seen even before the advent of antibiotics.

GEOTRICHOSIS. The *Geotrichum* has been recovered from 95 sputa, but the same difficulty arises in interpreting its significance as in monilia.

HISTOPLASMOSIS. Two cases of the virulent systemic infection were seen during the period under consideration. One presented with ulcers of the tongue and one with ulcers of the epiglottis and palate. Both were fatal.

The true incidence of Benign Histoplasmosis in this country is not yet established. Lurie [5] found 12% positive histoplasmin skin-test reactions in subjects most of whom were members of the staff of this Institute and could therefore have come into direct contact with the fungus. In a control group of 25 subjects in the Serum Laboratory of this Institute housed in a separate building no positive reactions were found. Jackson [6] found only 3 positive reactions in 453 subjects tested. However, Murray [7] while investigating a group of 23 speleologists who had suffered from 'cave disease' found 93% positive reactions. On the other hand a control group of 34 students in the same age-group as the speleologists showed no positive reactions. It seems certain therefore that, however the disease is contracted, Benign Histoplasmosis does occur in this country.

ASPERGILLOSIS. One case of pulmonary Aspergillosis has been found at autopsy and one was proved by bronchial biopsy. Two further cases were suspected from cultural examination of sputa but were not proven.

MUCORMYCOSIS. One case of Mucormycosis of the central nervous system was suspected on a histological section of post-mortem material but, as is usual in such cases, no fresh tissue was available for culture.

The incidence of the various diseases is summarized in Table II.

As yet no cases of North American Blastomycosis, South American Blastomycosis or Coccidioidomycosis have been encountered by the author. In more than 250 subjects selected at random and in several cases of undiagnosed pulmonary disease no positive coccidioidin skin tests were found.

SUMMARY

1. The incidence of various mycoses in South Africa based on the specimens examined at the South African Institute for Medical Research during the past 8 years is given. The results are summarized in Table II

2. The relative frequency of the various dermatophytes is summarized in Table I.

3. No cases of North American Blastomycosis, South American Blastomycosis or Coccidioidomycosis were encountered.

My thanks are due to the heads of the Departments of Pathology and Bacteriology of the Institute for access to their records.

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PRESENTATION OF BRONZE BUST OF PROFESSOR C. F. M. SAINT

A ceremony, unique in being probably the first of its kind in the history of South African Universities, took place in the newly erected C. F. M. Saint Museum of Surgical Pathology of the Cape Town Medical School, on 28 January 1955, when a bronze bust of Professor Saint, beautifully executed by H. H. von Michaelis, and commissioned by ex-house surgeons of Professor Saint, was presented to the University of Cape Town. A photograph of the bust is here reproduced.

About 50 people were present at the ceremony, the majority consisting of ex-house surgeons who are now practising in and around Cape Town; Mr. C. A. R. Schulenberg of Pretoria and Dr. A. G. Blyth of Ladismith, Cape, had however come to Cape Town specially for the occasion. Others sent messages of good will. The gathering also included other former colleagues of Professor Saint, and also Mr. von Michaelis.

Mr. W. G. Schulze (Hon. Secretary of the Committee) explained that the bust of Professor Saint, which was to be placed in the new Surgical Museum, was a special contribution from those who had been closely associated with Professor Saint either academically or professionally and, in particular, the house surgeons who had had the great fortune and privilege of working under him. During the period 1920-1946, when Professor Saint held the Chair of Surgery at the University of Cape Town, no less than 72 men and women had enjoyed this distinction. When it came to asking for contributions, there had been an almost 100% response, there being only one previous house surgeon (still living) who could not be traced. Eight had died. These contributions had come from all parts of South Africa, the Rhodesian Federation, the United Kingdom and the United States of America.

Mr. F. D. du Toit van Zyl (Chairman of the Committee) then spoke. He explained that when Professor Saint retired from the Chair of Surgery in 1946 he was presented with a gift of £2,500 from his past students. To this sum Professor Saint added an equal amount and donated the total to the University to form a nucleus for the building of a new Surgical Pathology Museum. With a £1 for £1 contribution from the State the sum now available was £10,000, and this sum has now been utilized in the erection of the present Museum, which is named after Professor Saint.

Mr. van Zyl spoke of Professor Saint as a great teacher, an eminent surgeon and a beloved chief. As a teacher he put forward the great principles on which Surgery was based in a way no other teacher they had known was able to do—principles which had been of life-long value in helping them to master the problems with which they had been faced.

Saint had come to this country as a young man. His fame rapidly

spread and he elevated surgery to a level undreamt of before in this country. This he had achieved by energy and example, by doing not by talking or criticizing others. South Africa would ever remain grateful to one whose service to his country had been as great as that of any hero of war. His devotion and ability had inspired a generation of men and women.

He was primarily a clinician—if anyone had a clinical sense he had it. But surgeons were made, not born; his clinical sense was the product of diligence. By the gentlest handling of tissues he avoided sepsis, in the days before antibiotics, and by speed he avoided thrombosis; and he minimized loss of blood in the days before massive transfusion. Yet he was bold and adventurous, though never reckless. His courage was conspicuous. Once in the presence of eminent onlookers he was operating on a very adherent and haemorrhagic spleen. He decided it was not safe to continue. He left the spleen with dozens of forceps on bleeding points—and successfully removed it a week later. He had the courage and initiative to retire from practice before his powers had declined.

Saint accepted his patient as an honour and with gratitude, whether a rich man or one from the slums. Never would he betray his trust or put his patient in jeopardy.

After mentioning Saint's international reputation—he had received honorary degrees in America and Australia—the speaker referred to his human side as a teacher. How meticulous he was over details, how loyal to subordinates! His loyalty to them created loyalty on their part. They were now no longer young; they had sat at the feet of other surgeons of world renown, and with their own maturity and experience they found that Saint's stature ever grew and their respect for him increased.

Because of these feelings they desired that his memory should be perpetuated. He had created a surgical tradition here as high as anywhere in the world; he had done great work for South Africa, for the University of Cape Town, and for the poor and the sick.

Mr. van Zyl then handed over the bust to the safekeeping of the University.

Dr. T. B. Davie, Principal and Vice-Chancellor, in accepting the bust for the University, said that it was a pleasant task to receive this tribute to a great man. He referred to the work of the 'triumvirate' Falconer, Saint and Crichton in fashioning the clinical side of the medical school. He, the speaker, had helped in the establishment of two museums of surgical pathology. He spoke of the function of such a museum in making the surgeon familiar with the naked-eye conditions he would encounter on the operating table. Surgery needed its own institute, with ready access to the physiologist, the pathologist and the radiologist.

Professor Saint in his reply remarked that surely few surgeons were heroes to their own house surgeons. He referred to the friendship and cooperation which existed between himself and his house surgeons and said his memory of all of them had been one of the greatest pleasures of his life.

ASSOCIATION NEWS : VERENIGINGSNUUS

CAPE MIDLAND BRANCH: ANNUAL MEETING

The annual meeting of the Branch was held on 20 January 1955 at the Provincial Hospital, Port Elizabeth. The result of the election of office-bearers was announced as follows:

President: Mr. H. I. Maister. *President Elect:* Dr. J. C. Rabie. *Vice-President:* Dr. M. A. Robertson. *Treasurer:* Dr. M. G. Woolff. *Secretary:* Dr. P. Jabkovitz. *Members of Branch Council:* Drs. A. P. Albert, J. R. McCoy, L. W. Finnemore, A. S. Weir, and J. A. Tarlie. *Ex-officio Members of Branch Council* are Mr. E. Colley, Dr. L. E. Lane and Dr. D. Ferguson.

Mr. E. Colley, the outgoing President, then inducted Mr. H. I. Maister as President of the Branch, who thanked the outgoing President for his services during the year. Mr. Colley then delivered his presidential address.

Mr. Colley began with a review of the events of 1954 and some reflections on them. He mentioned that the highlight of the year was the successful Medical Congress that was held in Port Elizabeth; other important events were the inauguration of the Branch's medical library and the opening of the Livingstone Hospital for the reception of patients. The Livingstone Hospital had a new series of problems peculiar to its site, such as the hordes of out-patients who descend on the hospital daily and are prepared to wait all day for treatment. This is a drain on the services of the hospital staff, including the honoraries.

He then went on to discuss the honorary system and pointed out that the hospitals of the Cape Province, excluding Groote Schuur, were the last stronghold of the honorary system in South Africa, and possibly in the Empire. The conditions under which the honoraries work might have to be reviewed *and the directions should not come from quarters where the honorary system had been abandoned.*

The services of the honorary staff would be extended to the full if the Livingstone Hospital was to function properly. This work would be hampered if the Livingstone Hospital were used to accommodate the chronic sick. These should be cared for by nursing aids and housed in rondavels on a small farm in the vicinity under conditions corresponding to their normal living conditions. In this way acute hospital beds could be saved costing £2,000 each.

The President mentioned that there appeared to be some dissatisfaction with the difficulties experienced by middle-aged general practitioners who wish to be registered as specialists. It would be a worthy effort for the General Practitioners Group to get some of the regulations altered by the Medical Council.

Mr. Colley then gave an analysis of the operations performed at the hospitals in Uitenhage and Port Elizabeth. The European population was 112,000, Coloured and Asiatics 72,000, and Africans 147,000. There were between 11,000 and 12,000 anaesthetics administered in a year and 3 deaths which could be directly attributed to the anaesthetic. The figures showed the remarkable frequency of certain types of operations and the rarity of others, and the marked disparity between the various racial groups. With the non-Europeans, who are exposed to the trauma of the hardest labour, operations for hernia, torn cartilage, varicose veins and slipped disc were infrequent, compared with these operations in Europeans. Operations for stab wounds were quite frequent and were the monopoly of the non-Europeans. A study of the figures might give much interesting information.

The President concluded by thanking the Medical Superintendent of the Provincial Hospital for housing the library and allowing the Association to hold its clinical and Branch meetings in the hospital environs, and paid tribute to the nursing staff for their kind hospitality.

The increase in the fees for Workmen's Compensation Act cases was noted, and the debt that the Association owes to the W.C.A. Committee of Federal Council was wholeheartedly endorsed.

It was further noted that the Leather Industry Sick Fund, after prolonged negotiations, had agreed to increase their capitation fees to that required by the Association. A difficulty still remained to be ironed out in that this Sick Fund was reluctant on general principles to pay a different capitation fee for Europeans and non-Europeans, although they had no objection to raising their fees to meet the mean requirements.

PASSING EVENTS : IN DIE VERBYGAAN

The wedding of Joyce, daughter of Mr. and Mrs. J. Fenster, Johannesburg, and Dr. Woolf (Willie) Klein, of Port Elizabeth, took place in Johannesburg on 6 February 1955.

Mr. and Mrs. Klein are expected back in Port Elizabeth on 21 February and will be staying at the Hotel Elizabeth.

Dr. C. K. O'Malley, 501-502 Geneva House, Church Square, specialist in venereology, has been registered by the South African Medical and Dental Council as a specialist in dermatology.

* * *

POLIOMYELITIS IN THE UNION

Following are the returns, supplied by the Union Department of Health, of cases notified under the Public Health Act as suffering from Poliomyelitis during the period 28 January to 3 February 1955:

	European	*Non-European*
Transvaal:		
Vereeniging	1	1
Johannesburg (1 fatal case)	6	3
Randfontein		2
Klerksdorp	1	
Pretoria	2	
Pretoria P.U.A.H.B.	1	
Silverton (fatal)	1	
Edenvale	1	
Vanderbijlpark	3	
Nylstroom		2
Springs	1	
Springs P.U.A.H.B.	1	
Witrivier district	1	
Krugersdorp P.U.A.H.B.	1	
Witbank P.U.A.H.B.		1
Oberholtzer P.U.A.H.B.	1	
Roodepoort P.U.A.H.B.	1	
Elsburg	1	
Kempton Park	1	
Potgietersrust	1	
Lydenburg	1	
Total for Transvaal	26	9
Cape Province:		
Clanwilliam Divisional Council		1
Cradock Divisional Council		1
Stutterheim Municipality	1	
Kimberley district		1
Cape Town	1	
Stellenbosch Divisional Council		1
Total for Cape Province	2	4
Natal:		
Ladysmith		1
Durban	1	2
Scottburgh		1
Umlazi district		2
Felixton	1	
Pinetown district		1
Pietermaritzburg district		1
Gillitts	1	
Drummond		1
Ndwedwe district		1
Marian-hill (fatal)		1
Kearsney		1
Stanger district	1	
Umbumbulu Mission	1	
Muden		2
Total for Natal	5	14
Orange Free State:		
Vrede		1
Total for O.F.S.		1
TOTAL FOR UNION	**33**	**28**

PARLEMENTÊRE NUUS

DIE NATURELLE (STADSGEBIEDE) WYSIGINGSWETSONTWERP

Deur 'n Parlementêre Beriggewer

Gedurende die debat in die Volksraad oor die tweede lesing van die Naturelle (Stadsgebiede) Wysigingswetsontwerp het 'n interessante bespreking van sekere mediese aspekte plaasgevind, met betrekking tot die teenwoordigheid van naturelle-bediendes op die persele van woonstelle of hotels. Die bespreking is gevoer deur twee mediese praktisyns, dr. C. de Wet, L.V. vir Vereeniging-distrik, en dr. J. van A. Steytler, L.V. vir Queenstown.

Dr. De Wet het o.m. gesê: „Ek wil my graag net bepaal by die sogenaamde lokasies in die lug .... Die agbare Minister (van Naturellesake) het hier gesê dat hy voorsien dat dit mag gebeur dat op 'n vierkante myl daar tot 30,000 van hierdie naturelle sal wees, in die jare wat kom ....

„Nie net in die nag nie, maar ook in die dag kry jy naturelle daar wat daar slaap. Met ander woorde, jy kry van daardie plekke bo-op die woonstelle waar daar dubbele bewoning plaasvind. Nou kry jy by die dubbele bewoning geen ventilasie van die plekke nie. Die deure is nooit oop nie. Daardie kamer is nooit vir een oomblik leeg nie, want in die dag kom daar van die naturelle wat in die nag gewerk het en hulle kom daar slaap.

„Nou kan u u voorstel watter ongewenste toestand en euwel dit is uit 'n gesondheidsoogpunt dat daar nooit sonlig in die kamer kom nie en dat daar nooit ventilasie is nie. Ek meen dat dit miskien een van die grootste oorsake is dat ons vandag die gevreesde epidemie van polio het, omdat die naturelle wat daar woon .... môre jou kinders moet gaan oppas en hanteer ....

„Waarom word daar gesondheidsvereistes gestel ten opsigte van die blankes, terwyl daar, as dit kom by hierdie nie-blankes, daaraan geen aandag gegee word nie .... Dit is 'n geweldige gevaar vir die blankes en die nie-blankes uit die oogpunt van die verspreiding van kieme as daar geen sonlig ooit in die vertrekke kom nie ....

„'n Vriend het my uitgenooi net voordat die parlementsitting begin het om spesiaal te gaan kyk in die Hillbrowwyk. Ek het een latrine daar gesien wat seker deur 400 of 500 naturelle gebruik word. Dink net aan die gevare. Dink net aan maagkoors of kinderverlamming. Wie sê dat dit nie deur water en so meer versprei word nie? Ons weet dit nie vandag nie. Maar die siektes waarvan ons weet hoe dit versprei word is erg genoeg om te sê dat ons die soort dinge nie kan toelaat nie ....

„Watter beheer kan 'n mens onder bestaande toestande uitoefen oor die verspreiding van siektes .... Sê daar breek 'n epidemie uit .... Hoe kan 'n mens 'n naturel wat daar siek word afsonder? .... As 30,000 naturelle daar kan saamdrom, dan moet daar tog ten minste op elke dag van die jaar 300 van hulle—op een vierkante myl wat deur blankes bewoon word—siek wees, siek in die bed. Hoe moet hulle daar versorg word? ....

„Wie is vir hulle verantwoordelik en wie moet hulle versorg? .... As ons dit bereken tot in die kleinste besonderhede, dan is dit maar 'n baie klein area waar daardie 30,000 nie-blankes moet woon ....

„As hierdie wetgewing aangeneem word, dan kan ons oorbewoning bekamp en sorg dat daar ordentlike en genoegsame voorsiening gemaak word .... Ek dink dat die mense wat dit die meeste sal verwelkom die mense is wat werksaam is in die Gesondheidsdepartement van die Stadsraad van Johannesburg."

Dr. Steytler het as volg op dr. De Wet se toespraak gereageer: „Dit is die eerste maal dat ek hoor dat enige medikus die isoleer van 'n aansteeklike geval toepas in die vertrek waar daardie geval voorkom .... As hy my kan oortuig dat daardie oortollige mense sal verskuif word na areas waar beter gesondheidsdienste geskep word, dan gun ek hom daardie punt ....

„Dit is ook die eerste maal dat ek as medikus hoor dat aansteeklike siektes, of die kieme wat verantwoordelik is daarvoor, beperk word deur afstand. As 'n pasiënt wat 'n aansteeklike siekte het 'n myl of twee myl verwyder word van die werkplek tussen die

blankes, dan is dit nog 'n kwessie of die beperking van daardie siekte daardeur bewerkstellig sal word."

TUBERCULOSIS IN THE UNION

During last year 30,364 cases of tuberculosis in all forms were notified in the Union, Mr. J. F. Naudé, Minister of Health, said in the House of Assembly last week (February 11) in reply to a question put by Dr. J. van A. Steytler, M.P. for Queenstown.

He added that the number of persons between the ages of 15 and 35 years who died of tuberculosis from 1944 to 1952 was 40,141. Mortality statistics beyond that date were not yet available. The figure was influenced, however, by the fact that up to June 1952 the registration of deaths of Natives, in terms of the Births, Marriages and Death Registration Act, No. 17 of 1923, as amended, was not compulsory in rural areas.

BOOK REVIEWS : BOEKRESENSIES

PROTEIN MALNUTRITION

Malnutrition in African Mothers, Infants and Young Children. Report of the Second Inter-African Conference on Nutrition, held under the auspices of the Commission for Technical Co-operation in Africa South of the Sahara (CCTA) at Fajara, Gambia, 19-27 November 1952. Published August 1954, London: Her Majesty's Stationery Office and agents, £1 5s.

This report is the record of a conference held in 1952, under the aegis of the British Government on behalf of the Commission for Technical Co-operation in Africa South of the Sahara (CCTA) at the Medical Research Council's Field Research Laboratory, Fajara, Gambia.

It is welcomed as a source of much valuable reference material but it is regretted that its appearance has been so long delayed. It can very profitably be read in conjunction with the report on the third session of the Joint FAO/WHO Expert Committee on Nutrition.[1] This session was held immediately after the CCTA Conference at Fajara, Gambia, and the members of the Joint Committee all attended the CCTA meetings. It is useful to know that it was in this context that the Joint Committee drew up the definition of 'protein malnutrition' which has had considerable publicity and which in turn became the subject of a conference in Jamaica in November 1953.

The CCTA Conference inevitably revolved around the problem of kwashiorkor and there were sessions covering this and related problems of infant malnutrition on (1) Clinical Features, Demography and Nosography; (2) Pathology; (3) Bio-Chemistry and (4) Dietetics. The final session was held under the title 'Malnutrition in mothers; and treatment and prevention of malnutrition in infants and children'. A section of the report is devoted to terminology and in an appendix compiled by M. Autret 36 names are given which have been used in various parts of the world for protein malnutrition syndromes identical with or closely related to kwashiorkor. In a footnote to this appendix the Editor of the CCTA report comments that the term 'protein malnutrition' might become generally acceptable. The subsequent third session of the Joint Committee proceeded therefore to define the term (page 5, paragraph 2.2).

Another useful appendix in the report is by H. C. Trowell. It includes provisional definitions of severe kwashiorkor and mild kwashiorkor.

In spite of its late appearance this report is a useful reference for those who are interested in problems of protein malnutrition and kwashiokor, and in the public health approach to widespread malnutrition.

J.F.B.

1. World Health Organization Technical Report Series No. 72, December 1953.

BOOKS RECEIVED : BOEKE ONTVANG

Handbook of Medical Treatment. Miscellaneous Authors, edited by M. J. Chatton, A.B., M.D., S. Margen, M.A., M.D., H. D. Brainerd, A.B., M.D. 4th edition. (Pp. 569. $3.00.) California: Lange Medical Publications. 1954.

Pathogenesis of Cancer. By John E. Gregory, M.D. 2nd edition. (Pp. 202 with illustrations. $7.50.) California: Fremont Foundation Publishers. 1954.

Fundamentals of Internal Medicine. By Wallace Mason Yater, A.B., M.D., M.S., F.A.C.P. 4th edition. (Pp. 1276 + xxx with illustrations.) New York: Appleton-Century-Crofts, Inc. 1954.

Surgical Progress. 1954. By Sir Ernest Rock Carling, L.L.D., F.R.C.S., F.R.C.P., F.F.R. and Sir James Paterson Ross, K.C.V.O., M.S., F.R.C.S. (Pp. 348 + vii with illustrations.) London: Butterworth & Co., Ltd., Medical Publications. 1954.

Hey Groves' Synopsis of Surgery. Edited by Sir Cecil P. G. Wakeley, Bt., K.B.E., C.B., D.Sc., F.R.C.S. 14th edition. (Pp. 651 + viii with 190 illustrations. 30s.) Bristol: John Wright & Sons, Ltd., 1954.

CORRESPONDENCE : BRIEWERUBRIEK

THE NATIONAL CANCER ASSOCIATION'S CAMPAIGN

To the Editor: The Executive of the Radiological Society of South Africa deplores and condemns the manner and matter of the contribution on the above subject in your *Journal* of 11 December 1954.[1]

The Executive is compelled to take up this matter because of the harm the article has already done in the Platteland and which it may still do, unless it is refuted, among the junior members of the profession, whether in Johannesburg or elsewhere.

The contribution contains, for instance, an extraordinary and erroneous version of a broadcast talk given by a radiologist at the request of the National Cancer Association Campaign Appeal Committee. It also shows ignorance of the organization of radiotherapy services discussed and advocated for many years by the Radiological Society of South Africa.

The article implies a superiority of the X-ray department at the Johannesburg General Hospital as shown by such statements as: 'The danger of a multiplicity of smaller centres within a single geographical region, is exemplified by the following avoidable tragedies on record in our files'; and 'Unfortunately, mismanagement of curable cancer is already all too frequent in both hospital and private practice in South Africa'. The writer, obviously, does not include the Johannesburg General Hospital in this condemnation.

The Executive feels that there is no justification for the condemnation of the efficiency of radiotherapy by private practitioners and X-ray departments at hospitals other than the Johannesburg General Hospital. It should be noted that these allegations of mismanagement are not made by the Johannesburg hospital authorities or by the Radiotherapist in charge at the Johannesburg General Hospital.

The Executive cannot condemn too strongly the derogatory statements made about the author's colleagues—both the clinicians on the staff of the Johannesburg General Hospital and elsewhere and radiologists. These statements accuse them of 'malpractice' and being influenced by considerations of 'bad business', merely because the author of the article does not agree with their views on the dosage necessary under various conditions. The following few examples will indicate how misleading the article is:

In the 3rd paragraph under the heading 'Regional Centres' on page 1065 there is a discussion of a broadcast by a 'radiologist' in the recent cancer campaign. The Executive of the Radiological Society has obtained the script of this broadcast and after careful comparison it states most emphatically that there is no resemblance between the script of the broadcast and the version of the broadcast

given in this article. There is no mention in the script of part-time radiologists, of the radiotherapy services being financed by the Cancer Association or of the establishment of multiple rural centres, which is so alarming to the author of the article.

The author's suggestions for the organization of the radiotherapy services in South Africa contain nothing new. They merely re-state incompletely the evidence given by the Transvaal Branch of the Radiological Society of South Africa in a memorandum submitted to the Gluckman Commission on 12 and 15 April 1943. The diagnostic and radiotherapy services for South Africa on a regional basis are fully discussed in this memorandum.

The author of the article admits that there are no statistics available of the efficiency of the treatment at various hospitals and in private practice. This being the case, he has no justification for condemning and passing derogatory remarks about outlying hospitals and radiotherapy in private practice.

The Executive does not accept the implication that radiotherapy at the Johannesburg General Hospital is superior to that in all other hospitals or in private practice, or that the Johannesburg General Hospital has facilities in apparatus or means of estimating dosage not available in private practice or elsewhere.

This is not the place to discuss the value of cancer detection schemes, but we would point out that the Chairman of the National Cancer Association, Dr. Robertson, in a statement to the Press recently declared that it was not the intention of the Cancer Association to put up cancer detection centres.

The Executive cannot therefore understand why the author of the article should enter into a totally irrelevant discussion of the subject and, moreover, take the results of a chest survey in Boston as typical of the results obtained in the Cancer Detection Centres in the United States. He does not give the reference on which his allegation was based. It would have been more reasonable to quote the incidence of new cancer cases found by the Cancer Detection Centres and published by them[2]. These figures vary (according to the facilities available at each centre) from 0·5% to 1% of new cancer cases. They do not support the allegation of an incidence of 1 : 100,000 which he quotes. The American Cancer Society reported the discovery of 0·8% cancers and 10% pre-cancerous lesions in a mixed group of 51,728 symptomatic and asymptomatic patients seen in Detection Centres throughout the country.[3] It was found impossible to exclude some patients with symptoms from the Cancer Detection Centres, but according to some reports, this does not make any material difference.[4]

There are numerous other points to which the Executive takes exception but does not consider worth discussion. It feels that sufficient reasons have been given for condemning the article which, because it contains so many misleading statements and accusations of malpractice against colleagues (both clinical and radiological), was unworthy of publication.

The Executive condemns, also, the reference to the National Cancer Association's Appeal as an imposition on the South African public and cannot too strongly deplore the unwarranted generalizations about the radio-therapeutic services in the hospitals and in private practices throughout the country.

H. Jackson
Hon. Secretary

Council of the Radiological Society
of South Africa
318 Pan-Africa House
Troye Street
Johannesburg
31 January 1955

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2. Lombard, H. L. *et al.* (1951): New Engl. J. Med., **245**, 797.
3. National Cancer Conference, 1st Proceedings, pp. 270-290. New York: American Cancer Society.
4. Cliffton, E. E. *et al.* (1951): Surg. Gynec. Obstet., **93**, 719.

THE TONSILLECTOMY CONTROVERSY

To the Editor: It is not strange that tonsillectomy always triumphs on this battle-scarred ground. Like the periodic eruption of Vesuvius so the anti-tonsillectomy campaigns belch forth and, when the hot air has blown over, there is a return to normality.

Several years ago there was a similar outcry in London by Mr. Tubby Layton of Guy's Hospital for the preservation of lymphoid tissue of the naso-pharynx. Again the age-old weather-beaten pros and cons of tonsillectomy were flogged and battered about; and as usual the vociferous appeals for tonsil preservation held the field for a short time only. This was followed by the inevitable vigorous attack by all and sundry on the tonsils and adenoids that had been held back from surgery. Then a normal balance returned and the combatants on the two sides sank into oblivion once again till the next round.

Whatever has been said for or against tonsillectomy is not new, and the lay press always joins in the frolic. History is bound to repeat itself.

J. Fine

204 Medical Centre
Jeppe Street
Johannesburg
3 February 1955

A UNIVERSITY NUMBER OF THE *Journal*

To the Editor: May I suggest that you issue a special 'University Number' of your esteemed *Journal* devoted entirely to the interests of the various medical schools in this country?

There are now four established medical schools in South Africa and a fifth one will be opened in the near future. An account of their activities, type of training, description and photographs of buildings, historical associations, research work, contemplated extensions etc. would be of great interest not only to post-graduates but also in the international sphere with particular reference to other parts of Africa.

If such a number could be published once every five years, an excellent and historically useful record of the progress of our medical schools could be built up over the years to come.

I think that this idea will receive wide support and trust that you will be able to give it your favourable consideration.

J. Henson

'Three Pines'
Maclear Road
Claremont
7 February 1955

STINKBLAAR POISONING

To the Editor: I have been greatly interested in the article by Drubin and Cohen [1] in your issue for 29 January, but feel that one or two corrections in connection with their remarks on Stinkblaar poisoning are necessary.

The fruit is not a berry nor is it ingested by children, or for that matter by any other type of living creature, in mistake for edible fruits. The fruit actually is a very prickly, nay spiky, seed-pod which contains a large number of small black seeds. The seeds, despite the fact that they are not in the least attractive, are sometimes eaten in large quantities by small children. The resultant poisoning may be very severe and it is also on record that acute intestinal obstruction has followed impaction of a mass of swollen seeds in the intestines. The seed-pod is as disagreeable to the palate as the leaf.

The seed also crops up as a cause of accidental poisoning through its inclusion in foodstuffs, mostly in bread or in beans. There are in the *Journal* and its predecessors several records of such occurrences. In addition, the seeds are used in some schools as a form of initiation, the object being to produce the deliriant effects of the alkaloidal active principles. This of course is an extremely dangerous practice because there is no control of the actual dosage which the child receives.

The authors go on to state that the plant 'flourishes abundantly on unused plots and on open veld'. Actually the plant is a weed of the surroundings of man and it is very seldom found growing in the natural open veld. My experience has shown that the plant almost invariable is found on ground which has been disturbed by man and then neglected. Children who are poisoned by the plant, apart from the possibility of accidental bread poisoning with the seed, obtain the plant in the neighbourhood of their dwellings or play-places and, as stated by Drubin and Cohen, it is an easy matter to get rid of this source of danger to our children.

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1. Drubin, R. and Cohen, H. (1955): S. Afr. Med. J., **29**, 101.